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PARALYSES

CEREBRAL, BULBAR, AND SPINAL.

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PARALYSES

CEREBRAL BULBAR AND SPINAL

A MANUAL OF DIAGNOSIS

FOR

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BY

H. CHARLTON BASTIAN, M.A., M.D., F.R.S.

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PREFACE.

THIS work is intended as an aid to the student or medical practitioner when brought face to face with cases of paralysis of different kinds.

In special treatises all that is known of the several forms of paralysis may be set forth in the fullest manner. That is not what has been aimed at here. The author's endeavour throughout has been to facilitate diagnosis—to explain and gather up the essential points to be borne in mind by the student or practitioner when he is called upon to decide as to the nature of, and give a prognosis concerning, any case of paralysis that may come before him.

The various forms of paralysis from brain disease, from disease of the bulb, and from disease of the spinal cord are now so numerous, and so many recent advances have been made to our knowledge in these directions, that some such aid to diagnosis may well be looked for by those for whom this work is intended.

The signs of paralysis of the different cranial nerves have been pretty fully dealt with, because the recognition of such paralyses, either alone or in association with paralysis in other parts, is often a matter of the greatest importance.

A knowledge of nervous diseases of the kind now looked for cannot be attained without something more than a superficial acquaintance with the anatomy and physiology of the brain and spinal cord: hence the plenitude of anatomical details, more especially, which are absolutely essential in a work like the present. The comprehension and recollection of these have been facilitated, as far as possible, by the reproduction of a number of illustrations mostly borrowed from standard works.

Concerning the importance of arriving at a correct diagnosis it is needless to dilate. Without it, prognosis and treatment upon a scientific basis are alike impossible.

QUEEN ANNE STREET,
CAVENDISH SQUARE.
June 21st, 1886.

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PARALYSES, CEREBRAL, BULBAR, AND SPINAL:

A MANUAL OF DIAGNOSIS.

Paralysis and convulsions are opposite phenomena, the one dependent upon impeded conduction of motor stimuli to muscles, owing to some morbid conditions in or acting upon certain nerves, parts of the spinal cord, or encephalon; the other dependent upon an exalted transmission of motor stimuli to the muscles affected, owing to the existence of abnormal states of certain portions of the encephalon or spinal cord, but mostly of the former.

Thus paralysis is a sign of defective action, convulsion a result of exalted or perverted action in some portion of the nervous system.

In diseases of the encephalon convulsions often coexist with paralyses.

In morbid conditions of the spinal cord and of the peripheral nerves convulsions are but rarely met with, and when they occur they are only very indirect consequences of such conditions.

Paralyses of different kinds are, however, common and direct consequences not only of diseases of the encephalon, but also of the spinal cord and of peripheral nerves.

Although the word paralysis ought, strictly speaking, to be limited to the results of disabilities occurring in the parts of the nervous system where outgoing currents originate or through which they are transmitted, still custom has sanctioned the use of the same term as a designation for analogous defects resulting from functional disabilities in sensory nerves and sensory centres, or in the intervening tracks for such impressions.

We must, therefore, claim to be allowed to speak here of sensory paralyses as well as of motor paralyses. These two kinds of defect are often to some extent conjoined in the several forms of encephalic, spinal, and peripheral paralysis. The different modes and degrees of such association constitute some of the principal differentia by which the extremely varied forms of paralysis may often be distinguished from one another, as dependent upon defect in this or that part of the nervous system.

Paralyses are almost, if not quite, invariably caused by definite morbid conditions appreciable either by the naked eye or by the microscope, or by both.

Convulsions, on the other hand, are in all probability as invariably caused by mere molecular disturbances, inappreciable either by the naked eye or through the microscope. This view is not really contradicted by the fact that convulsions so often occur in association with, and as apparent consequences of, various organic diseases of the brain. The organic disease does not immediately give rise to the convulsion; it favours from time to time, through the intervention of other unnatural processes, disorderly molecular movements in certain of the nerve centres, which are the immediate forerunners of convulsions.

Neither of these positions would receive the assent of all pathologists and physicians. Some believe that paralyses not unfrequently exist without visible structural defects of any kind; whilst probably many could be found who would say that 'coarse' pathological lesions are often the actual causes of convulsions.

The causes of the morbid states with which we are now concerned —that is the different forms of paralyses—are to be found, therefore, occupying the widest possible range from the point of view of their kind or nature. They extend from mere molecular or nutritional perversions through the whole gamut of pathological conditions—the commonest of which are hæmorrhage, thrombosis, embolism, and new-growth.

Functional and structural morbid conditions of the cerebro-spinal nervous system have, therefore, alike to be considered in their relation to different kinds of paralysis. Instances of paralysis due to mere functional defects or perversions will, however, be found to be comparatively rare; and when existent will be sometimes cerebral but perhaps more frequently spinal in their type.

The diagnosis of diseases of the nervous system is now, by common admission, a two-fold problem. The locality as well as the nature of the lesion has as far as possible to be determined. In other words, a **Regional Diagnosis** as well as **Pathological Diagnosis** has to be arrived at.

It has been more especially during the last five and twenty years that the pathological and physiological knowledge has been accumulated which now in many cases makes the diagnosis of locality a possibility. Formerly only the outlines of such a diagnosis were attempted; at the present day something more is demanded. Formerly a pathological diagnosis combined with some such outline of a regional diagnosis was deemed sufficient, and accordingly 'softening of the brain,' 'cerebral hæmorrhage,' and such like terms, were considered not only as adequate diagnoses but as suitable headings under which to describe the results of the pathological conditions in question, almost irrespective of the frequently profound differences resulting from difference in site. Such a mode of describing brain diseases was at first inevitable by reason of the defective knowledge of the time. It survived perhaps longer than it need have done, and then became a drag to progress. Of late years vigorous efforts have been made—and not without a large measure of success—to put an end to this state of things. Now it is commonly recognized by pathologists and physicians that the brain is a congeries of many organs or parts, disease in which produces very dissimilar signs and symptoms; and, similarly, it is commonly recognized that the effects of disease occurring in different parts of the transverse area of the spinal cord should be carefully discriminated from one another. The recognition of the different results flowing from pathological changes affecting different longitudinal levels of the spinal cord was too obvious to have escaped early recognition, so that to this extent studies in localisation had been commonly attempted.

In fact the same kind of precision which has been long looked for in regard to the diagnosis of affections dependent upon disease or injury of any of the peripheral nerves, we now strive—however ineffectually in many cases—to import into the diagnosis of diseases of the encephalon and of the spinal cord.

In attempting to attain to some such precision as regards the **Regional Diagnosis** of encephalic and spinal diseases we are met by many difficulties. One fertile cause of difficulty is due to the fact that, beyond the particular signs and symptoms caused by destruction of some given portion of one of the great nerve centres or by irritation of the same region ('direct' effects), other complicating sets of effects are apt to be produced in a much more irregular manner. These latter complicating signs and symptoms may be brought about either in a simple mechanical manner through the agency of pressure, or by reason of physiological relationships between the regions affected and some more or less distant parts, as a consequence of which functional disturbances are caused in these parts, either stimulatory or inhibitory in their nature. These 'indirect' effects of cerebral or cerebellar lesions are sometimes more obvious and prominent than the 'direct' effects of such lesions. The practitioner of medicine, therefore, in his attempts to localise disease, must not be too strictly guided by the teachings of the physiologist. He has to endeavour to discriminate the particular combinations of signs and symptoms which appertain to lesions in different regions of the encephalon, however they may be induced. It is of comparatively little importance to the physician, so far as his purpose is concerned, whether any particular symptom is of 'direct' or of 'indirect' origin. His knowledge has been, in the main, slowly built up by himself and others through the careful study during life and after death of typical and uncomplicated cases of encephalic disease. The experiments of the physiologist on lower animals often suffices to sharpen the observation of the physician in detecting the signs of disease in man. They may, indeed, open his eyes to the existence of signs or symptoms previously unnoticed. But they do little more—we have ultimately to fall back upon ordinary clinical and pathological observations, in our attempts to determine the groups of signs and symptoms produced by disease in this or that region of the brain of man.

The variations met with in different cases of disease occurring in the same part of the encephalon owing to differences in the extent and suddenness of the lesion are almost, though not quite, as apt to affect the 'direct' as the 'indirect' symptoms. Like causes should produce like results—and this dictum ought, in the main, to hold good for the 'indirect' as well as for the 'direct' results of brain lesions.

Thus we, as practitioners of medicine, are in a measure independent of some of those difficult questions which at present perplex the cerebral physiologist—questions as to whether the symptoms in this or that cerebral lesion depend upon the abolition or stimulation of function of the part involved, or whether they are due to stimulatory or inhibitory effects upon functions of distant parts, brought about either by obscure reflex agencies, by unknown anatomical communications, or by pressure indirectly occasioned.

Under the head of **Pathological Diagnosis** we have to do with a distinct set of enquiries, and for the most part we rely upon evidence of a different character from that which guides us in determining a regional diagnosis. The most important question that presents itself under this head is, whether we have to do with a merely functional or with a structural disease. Still, as already intimated, this is a question which will not much concern us in reference to the great majority of cases of paralysis. Here we have, only too frequently, to do with structural disease of one kind or another.

The diagnosis of functional disease must always be arrived at by way of exclusion. We must first, by the most careful examination, satisfy ourselves as to the probable absence of all kinds of structural disease, before we have resort to such an explanation.

Structural diseases themselves are of many different kinds. Roughly speaking they are divisible into two categories, viz., acute and chronic. To the former category belong hæmorrhage, embolism or thrombosis (softening), and inflammation; to the latter, pachymeningitis (subacute or chronic), insular and band-like sclerosis, tumours, abscesses, aneurysms, and parasites. The immediate results of traumatisms would, as regards the production of paralysis, take rank in the former category; while the more remote effects of such injuries would find a place in the second category.

The sum total of signs and symptoms met with in encephalic or in spinal diseases is influenced much more profoundly, other things equal, by the locality of the lesion than it is by the nature of the lesion. In other words we should approach much more closely to a correct picture of the facts presented by the bedside study of disease, if we were to describe the results of destructive lesions in different regions of the great nerve centres and ignore the particular nature of the destructive lesions, than if, following more closely the method formerly pursued, we were to enter into elaborate descriptions of hæmorrhage into, or of softening of, the brain, without reference (or with only the briefest reference) to the great differences in the sum-total of symptoms as one or other of these pathological processes implicates this or that portion of the brain. From the point of view of symptomatology in fact, the region affected is of much greater significance than the nature of the lesion. Hence it might be thought that to solve the problem of regional diagnosis in any given case is a matter of much greater importance than to arrive at a correct pathological diagnosis. That is true only in part. It indubitably holds good, as above stated, in regard to symptomatology; but from the point of view of prognosis and treatment, in the large majority of cases, it is of distinctly greater importance to be able to make a correct pathological diagnosis.

Not for these reasons only, but also because of the nature of the considerations which are in the main taken into account in attempting to arrive at a pathological and at a regional diagnosis respectively, precedence should be given to the questions concerning pathological diagnosis, when we are studying diseases of encephalic origin, though in the case of paralysis of spinal and of peripheric origin it will be found most desirable to reverse the process and deal first with the question of regional diagnosis. The adoption of the former method in investigating paralyses of encephalic origin is rendered advisable by the fact that in the brain our knowledge of the functions of its several parts is even less definite than the corresponding knowledge in regard to the spinal cord, and also because in the brain the several kinds of pathological changes are less apt to be met with only in particular regions of the organ.

In seeking to arrive at a pathological diagnosis we must concentrate our attention upon the following points :—The age, sex, and family history of the patient ; his past personal history, including diseases and accidents experienced, occupation, habits and mode of life ; his state of nutrition generally, and especially the condition of his heart and arterial system ; the date of the first signs of his present illness, together with its rate and mode of increase ; the precise nature of the signs and symptoms presented at the onset of the patient's illness and at subsequent stages. Information concerning such facts must be supplemented by a thorough examination of this condition in regard to nervous functions at the time of his coming under observation (which will often be not during the first stages of his illness). All such data have to be taken into account ; and then the net-total of resulting information has to be mentally compared with what we know concerning the several possible pathological causes of such kinds of disease in order that we may judge which of them has been, or has most probably been, operative in the case under consideration. Occasionally it happens, and this is more especially the case in regard to certain spinal paralyses, that the problem of pathological diagnosis is very much simplified, and also that it may be settled in the main from a mere study of the patient's present condition—that is from the characteristics of the established disease, coupled with some information as to its rate and mode of onset.

In seeking to arrive at a regional diagnosis we must be guided in the main by the results of the fullest examination of the patient's actual condition as regards degree and range of motor paralysis; degree and distribution of sensory impairment, if any; degree and kind of impairment in the functions of one or other of the cranial nerves; integrity or the reverse of speech, and, if defective, the precise nature of the defect; impairment or not of deglutition; the mental condition of the patient; the degree of control over sphincters. The sum-total of results thus obtained must be viewed in the light of previous clinical experience, illuminated by our knowledge of the anatomy and physiology of the nervous system, and of the topography of its several parts.

Having arrived at a regional diagnosis in the manner above indicated, it is possible that this regional diagnosis may reflect some light upon the pathological diagnosis—because of the known proclivity of certain pathological conditions to occur in some rather than in other regions of the nervous system.

PRELIMINARY REGIONAL DIAGNOSIS.

Paralysis of any group of muscles—those of the limbs or elsewhere, and howsoever occasioned—may, of course, be complete or incomplete; and it may be as well here to add, that it is the incomplete forms of paralysis in the limbs (cases of 'paresis' as they are often termed) which are most apt to be confounded with certain weaknesses or motor defects due to joint disease. It is obviously a matter of first importance to be clear whether we have to do with a case of paralysis or not—and it may be laid down as a general rule, that the cases most likely to inspire a legitimate doubt on this subject will be those in which the paralysis, of whatsoever kind, is incomplete rather than complete.

It has long been customary to make a mere preliminary regional diagnosis in cases of paralysis—that is to decide in the first case what great division of the cerebro-spinal nervous system is in fault. This kind of preliminary diagnosis is usually very soon and very easily made by those who have been accustomed to observe cases of nervous disease; students, however, and some young practitioners occasionally experience difficulties in arriving at a positive opinion even on this preliminary problem.

It is necessary, therefore, briefly to consider paralysis in general, and the mode of distinguishing its primary divisions.

A loss of the power of voluntarily exciting the contraction of one or more muscles is the essential condition met with in all forms of motor paralysis. And, similarly, a loss of the power of transmitting impressions (either those of the special senses or those of common sensibility) from the several sites in which they take origin, inwards, or inwards and upwards, to those parts of the brain that are concerned with their realisation in consciousness is what is commonly known as 'sensory paralysis.'

For practical purposes the several kinds of paralysis are divisible primarily into three distinct categories, based upon the situation of the damage, lesion, or defective activity by which they are occasioned. We have, therefore :—

1. PARALYSES OF ENCEPHALIC ORIGIN,
2. PARALYSES OF SPINAL ORIGIN,
3. PARALYSES OF PERIPHERIC ORIGIN,

according as the cause is one which operates upon or within some part of the great centres within the cranium, upon or within some part of the spinal cord, or upon or within some one or more of the nerve trunks, in portions situated either inside or outside the cranio-spinal canal.

To which of these categories does the case before us belong? This is the primary or preliminary diagnosis which has to be made. In the great majority of instances it is capable of being made with great certainty. In considering it the student or practitioner must be guided by the more or less exact agreement of the signs and symptoms presented by the case before him, with one or other of the following combinations of signs and symptoms.

1. Paralyses of Encephalic Origin.—These may or may not be ushered in by an apoplectic attack, or by an epileptiform fit or a series of them. The paralysis is usually confined to one half of the body, though only certain parts of this are affected, namely, more or less of one half of the face, with the arm and the leg (either completely or incompletely) on the same side, while the muscles of the trunk are comparatively little affected. Where the paralysis is incomplete the arm is commonly more affected than the leg. Except where loss or impairment of consciousness still exists, or where both sides of the brain are affected, the patient almost invariably retains control over the bladder and rectum.

The common sensibility of the affected half of the body may for a shorter or longer period from the commencement of the illness be more or less diminished. The electrical irritability of the paralysed muscles is not notably altered. The superficial reflexes may be diminished, and the deep reflexes may be exalted on the paralysed side of the body.

These are the general characters of a form of paralysis commonly known as Hemiplegia. It is met with almost as frequently on the one as on the other side of the body, and may occasionally affect both sides simultaneously.

2. Paralyses of Spinal Origin.—These forms of paralysis almost invariably commence without impairment of consciousness or convulsions, though like those of the last category they may be either sudden or gradual in their mode of onset. They are, however, commonly characterised by their implication to a variable extent, of both sides of the body. In the great majority of cases the lower extremities, either alone or with the trunk muscles up to a certain level of nerve supply, are the parts that are paralysed. The arms are much less frequently affected. It is common for control over the bladder and rectum, one or both, to be more or less lost. The motor paralysis may exist with little or no impairment of sensibility; though in many cases sensation in one or other of its modes is defective in the paralysed parts. The upper limit of defective or altered sensibility is often marked round the trunk by a sense of constriction, or a feeling as if a band were tied round the body ('girdle sensation'). The electric irritability of the paralysed muscles may be either little altered, or it may be modified in the manner to be described subsequently (p. 10) as characteristic of the 'reaction of degeneration.' In these latter cases, early and marked atrophy of such paralysed muscles commonly ensues.

Many of the above forms of paralysis were long, and are still, commonly included under the common name, Paraplegia. Both sides of the body are usually affected—equally or unequally—because of the frequency with which the lesion, or cause of the disease, involves both halves of the spinal cord. Where this is not the case, and the injury or lesion is confined to one half of the cord in one or other region, a condition known as Hemiparaplegia results, in which, in addition to other special characters, there is a complete absence of facial paralysis, even though the arm and the leg on one side of the body (when the lesion exists high up in the cervical region of the cord) may be paralysed in much the same manner as they are in hemiplegia. In hemiparaplegia, however, the paralysis of motion occurs on the same side as the lesion or injury; whilst any sensory impairment that may exist occurs on the opposite side of the body.

3. Paralyses of Peripheric Origin.—The majority of paralyses resulting from disease or injury of motor nerve trunks are rendered comparatively easy of recognition by the fact that the loss of power is limited to muscles or groups of muscles supplied by particular nerves. This circumscribed nature of the paralysis is a fact of great value for diagnostic purposes—especially when the loss of power is complete rather than partial—because it is in these cases more particularly that we are apt to find another characteristic sign of peripheral paralysis, namely, an altered electrical excitability of both nerve and muscles. Where the injury to or disease of a nerve trunk is well marked, so that its fibres are either severed or rendered in any way incapable of conducting motor stimuli from the centres, it is found that within a period of 6-14 days the following electrical phenomena may be detected:—

1. Loss of irritability of the affected nerve-trunks to both Faradic and Voltaic currents.
2. Loss of, or greatly diminished, irritability of the affected muscles under stimulation by the Faradic current, together with an increase of their sensitiveness to the Voltaic current beyond that of healthy muscles.
3. Also a qualitative change in the irritability of the affected muscles to the Voltaic current in which the Anodal Closure Contraction (A.C.C.) is developed as easily or even more easily than the Cathodal Closure Contraction (C.C.C.); also that the Cathodal Opening Contraction (C.O.C.) generally becomes more easily developed than the Anodal Opening Contraction (A.O.C.).

These qualitative changes are the reverse of those exhibited by healthy muscles.

The electrical characteristics just cited constitute as a whole the so-called '*reaction of degeneration*.'

To the characters above mentioned must be added the further peculiarity that the muscles thus affected are apt speedily (within two or three weeks from the onset of the paralysis) to show a marked amount of atrophy—a change easily to be appreciated in limb muscles, and in some of those pertaining to the trunk, but by no means so obvious in the muscles of the face.

Paralyses resulting from disease of sensory nerves, special or other, are of course characterised by the nature of the special loss, or by the area over which common sensibility is impaired or lost in different cases.

Where we have to do with serious disease or damage to mixed motor and sensory nerves, this is detected by the recognition of motor and sensory defects so combined as to be compatible only with impairment of function in this or that mixed nerve.

The above constitute the characters which are in the main to be relied upon for the diagnosis of paralyses of peripheric origin. Still it must not be forgotten that when the ganglion cells in the spinal cord or medulla oblongata, which constitute the nerve-nuclei of the several motor nerves, are diseased, almost precisely the same effects are produced as if the nerve trunks had been damaged in some part of their course. We may have, that is, in each case, the electrical 'reaction of degeneration' followed by speedy atrophy of the affected muscles. Such diseases, of which acute spinal paralysis and glosso-laryngeal paralysis are examples, are always classed as centric paralyses. They are, it is true, spinal or bulbar in their seat; and yet they are attended by all the clinical characters pertaining to disease of the nerve trunks. This is natural enough seeing that the morbid process would, in fact, in such diseases as those above specified, simply affect the ganglion cells at the proximal extremities of nerve trunks, situated in the grey matter of the spinal cord or of the bulb.

In the present work we shall deal with the subject of Peripheral Paralysis only so far as it concerns the nerves that arise from the centres contained within the cranium.

PART I.

PARALYSES OF ENCEPHALIC ORIGIN.

PRELIMINARY DATA.

Certain preliminary anatomical data closely related to the problems involved both in the Pathological and in the Regional Diagnosis of encephalic diseases ought to be set forth, for future reference, before actually entering upon the study of these problems.

The information here briefly given, or conveyed by the figures, will afford a necessary anatomical basis concerning the Vascular Supply of the Brain, the Topography of the Cerebral Convolutions and Fissures, and also as to the Correspondence of the principal Fissures and Convolutions with definite external Regions of the Head.

These are subjects as to which an exact knowledge is now of the highest importance in reference to the diagnosis of Encephalic Diseases, as well as in making autopsies and recording the pathological results of such diseases. Occasionally, also, the particulars given under the last head will be found to be of importance in reference to some surgical operation that may be deemed advisable in certain rare cases of disease in the cerebral cortex.

VASCULAR SUPPLY OF THE BRAIN.

It would be superfluous here to describe in detail the source and mode of arrangement of the great arteries at the base of the brain. In this situation they enter into the well-known Circle of Willis, the normal arrangement of the vessels in which may be seen by reference to Fig. 1. It is well to bear in mind however that variations from this normal arrangement are by no means unfrequently met with. Variations in the size of the two posterior communicating arteries, and also in the relative size of the two vertebral arteries are especially prone to occur.

Our later and more exact knowledge in reference to the vascular supply of the brain has been due in the main to the independent and almost simultaneous investigations of Heubner and Duret.

Heubner divides the entire arterial supply of the encephalon into two systems—viz., a **basal** and a **cortical arterial system.**

Duret's primary division is essentialy the same, except that he uses the term 'central' to designate what Huebner speaks of as 'basal.'

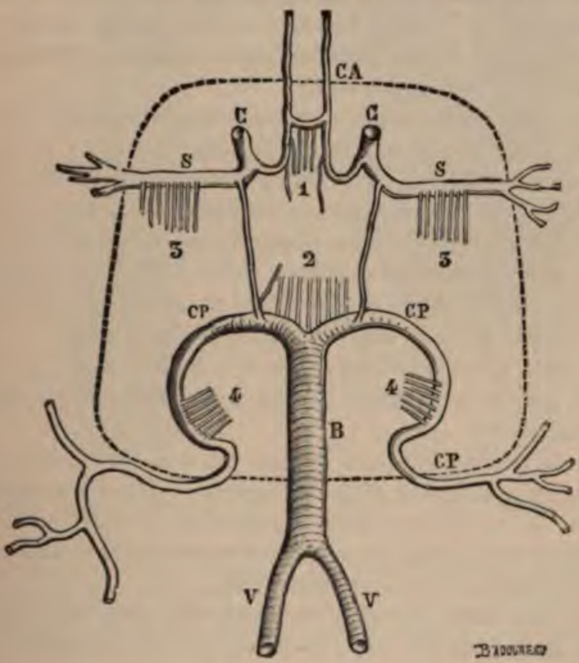

FIG. 1. DIAGRAM OF THE ARTERIAL CIRCULATION AT THE BASE OF THE ENCEPHALON [after Charcot].

C, C, Internal carotids. CA, Anterior cerebral arteries. S, S, Sylvian arteries V, V, Vertebrals. B, Basilar artery. CP, CP, Posterior cerebrals. 1, 2, 3, 3, 4, 4, groups of basal arteries. The dotted line shows the limits of the basal circle.

The **Basal Arterial System** is represented by the circle of Willis and the principal trunks of the cerebral arteries for a distance of ⅓-1″ from this circle. From these trunks numerous small or very small branches pass off at, or nearly at, a right angle and sink almost immediately into one or other of the important ganglia near the base of the brain. These vessels are true terminal or 'end' arteries which do not anastomose either with one another or with the vessels of the cortical arterial system. In Fig. 1 they are represented as forming six groups (1-4), the distribution of which will be considered further on.

The **Cortical Arterial System.** This system is composed of three large vessels on each side which come off from the circle of Willis, viz., 1, the anterior cerebral; 2, the middle cerebral (or Sylvian)—both of these being terminal branches of the internal carotid within the cranium; and, 3, the posterior cerebral, which is derived from the bifurcation of the basilar artery.

Together, these arteries are distributed over the whole surface of the brain, each having a pretty well defined territory of its own, which, however, is divided into departments corresponding with the distribution of the several secondary branches of each pair of primary trunks. The distribution of the vessels in these territories and departments we shall elsewhere describe in detail. We must first refer to certain characteristics common to all of them, and related to their mode of distribution over and through the cerebral cortex.

Each main trunk gives off from two to five named secondary branches, and each of these divides into two or three unnamed tertiary branchlets.

From the tertiary branchlets proceed on each side in the plain of the pia mater a multitude of fine filaments. Duret says these filaments do not anastomose with one another, though he believes a communication does take place to some extent between the terminations of the branchlets of contiguous areas.

In the pia mater therefore we have, (1) main arterial trunks, with (2) their branches, (3) branchlets, and (4) multitudes of fine filaments, the latter especially being distributed with the prolongations of the pia mater which dip into the sulci. From the third and fourth sets of vessels multitudes of minute arterial twigs pass inwards at right angles so as to penetrate the cortex over its whole area. These ultimate arterial twigs (of the 5th order) are commonly known as nutrient arteries; they are extremely slender and almost resemble capillaries, they penetrate the surface grey matter (in all parts of the convolutions and sulci) in a direction perpendicular to its surface. Some are short, others are long. The former are smaller than their fellows and terminate in capillary networks within the grey matter itself (Fig. 2). The longer twigs pass through the grey matter (giving offsets as they go which communicate with the capillary network) into the white substance beneath, penetrating the centrum oval for a distance of 3-4 centimètres (1¼″-1½″).

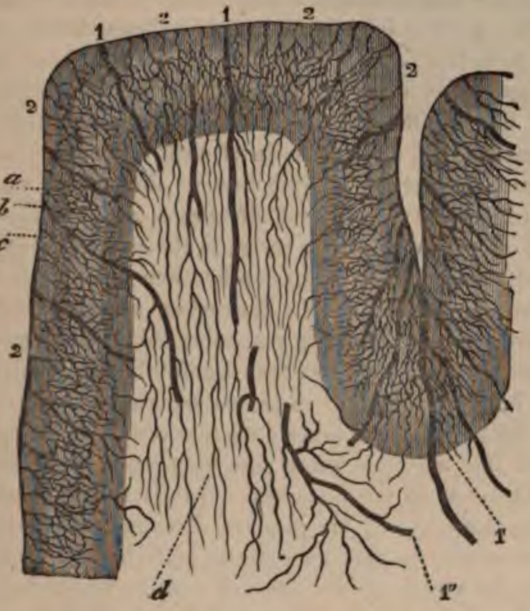

FIG. 2. PLAN OF DISTRIBUTION OF ARTERIES IN THE CEREBRAL CORTEX [after Duret].

1, 1, Long nutrient (or medullary) arteries; 1', ditto in the sulcus; 1'', ditto among Gratiolet's commissural fibres; 2, 2, 2, short nutrient (or cortical) arteries. *a*, capillary network with fairly wide meshes situated beneath the pia mater; *b*, network with more compact polygonal meshes situated within the cortex; *c*, transitional network with wider meshes; *d*, capillary network in the white matter.

Towards the boundary of their area of distribution these nutrient twigs of the cortical system approach the terminal twigs of the basal arterial system. But the two sets of vessels are said not to communicate with one another.

From the above description it is evident that in cases of thrombosis or embolism occurring in vessels of the cortical system, the grey matter of the cortex and the subjacent white matter are apt to suffer together—seeing that they derive their blood supply from the same nutrient arteries. The size of the patch of superficial softening must of course depend in part upon the size of the vessel obliterated; though it would also be largely influenced by the amount and freedom of the anastomoses, if any, existing between the terminal ramifications of this vessel and those of its neighbours.

In regard to the question as to the degree of anastomosis existing between the vessels of the cortical system, opinions differ. Heubner believes it to be free between the different main vessels of the cortex and also between the secondary branches, basing his opinion upon the results of his injections. This seems to be undoubtedly true in certain persons; and the existence of such a condition in a case in which there is obstruction by embolism or thrombosis, would permit the establishment of a collateral circulation, and, consequently, the staving off of softening of the cortex in the area corresponding with the blocked vessel. Such an exemption from softening undoubtedly occurs in certain cases of arterial obstruction, as post mortem examinations have shown. Heubner maintains that the anastomoses are effected through vessels not less than a millimètre in diameter.

Duret, however, contends that anastomoses between the main trunks or the secondary branches are either absent or extremely rare. He thinks that communications are only brought about through terminal filaments of the branchlets—that is through vessels which are not more than $\frac{1}{100}''$—$\frac{1}{120}''$ in diameter. Even such communications he believes are variable in number in different individuals. When they are pretty free, the vessels of the different vascular territories or departments communicate with one another at their periphery; and for this reason when there is obliteration of a main trunk, softening is either limited to, or much more obvious in, the central regions of the vascular area.

Cohnheim also believes that the cerebral arteries which are not true terminal or end arteries approximate closely to this type—that is, that they have no communications with surrounding vessels except through their ultimate capillary loops. Obliteration of any such vessel must lead to the death (and consequent softening) of the tissues in all but the peripheral parts of the area which it supplies. If a collateral circulation could be established such death or softening of tissue would be avoided, and temporary functional disturbance would be the principal result of the occlusion.

Details concerning the distribution of the different vessels belonging to the cortical and basal systems will be found in the part devoted to Regional Diagnosis, prefatory to a consideration of the effects of lesions in the different Encephalic Regions.

RELATION OF THE PRINCIPAL FISSURES AND CONVOLUTIONS OF THE CEREBRUM TO THE OUTER SURFACE OF THE SCALP.

Several observers have made a careful study of the relations existing between the principal sulci and convolutions and the various fissures of the bony cranium, but as these sutures are frequently not to be felt through the scalp such relations are of little value to any one examining the head from a medical or a surgical point of view. The realisation of this fact induced Dr. R. W. Reid to make a careful study of the relations of the principal sulci to the outer surface of the scalp.

The two figures which are here reproduced (Figs. 3 and 4), together with the following abstract of his results give, therefore, information which may be of real practical value. Reid says his method "has been frequently tested on the dead body and found to be accurate, and is merely intended to afford, for practical purposes, a general idea of the surface of the cerebrum in relation to the scalp." It does not take into consideration the shape of the head in different races, or in those individuals whose skulls present an abnormal outline owing to pathological conditions.

Absolute accuracy cannot be expected from any such method, for the simple reason that the sulci are never in any two brains precisely alike, or the shape of the head exactly the same in all cases. Again it must be remembered that, "as the surface area of the outside of the scalp is greater than the surface area of the corresponding part of the cerebrum, any lines drawn on the scalp to represent the sulci will be longer than the sulci themselves, and the spaces between those lines indicating the convolutions, will be broader than the convolutions themselves." Still, Reid shows that by taking large and easily felt landmarks on the head, and drawing from these certain lines, such lines will indicate accurately enough for all practical purposes the position of the principal sulci.

The base line, from which all perpendicular lines are drawn, runs through the lowest part of the infra-orbital margin and the middle of the external auditory meatus (Fig. 3).

The situation of the *longitudinal fissure* needs no description, whilst that of the *transverse fissure* is indicated by drawing a line along the superior curved line of the occipital bone, from the external occipital protuberance to the middle of the external meatus.

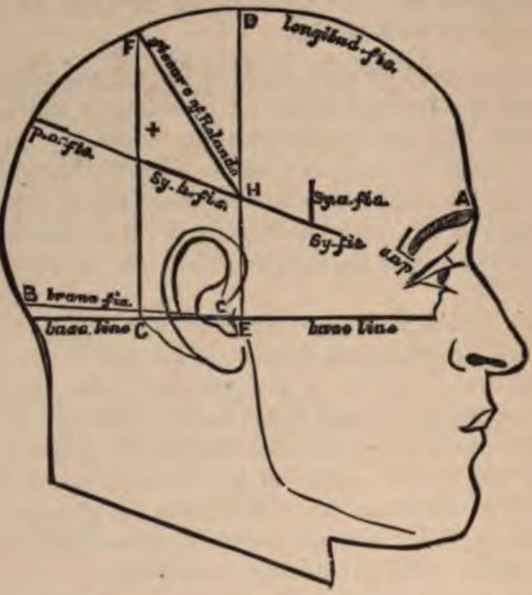

FIG. 3. DIAGRAM ILLUSTRATING THE POSITION OF THE PRINCIPAL CEREBRAL FISSURES RELATIVE TO THE SURFACE OF THE SCALP [after Reid].

A, Glabella. B, External occipital protuberance. eap, External angular process of the frontal bone. BC, Transverse fissure. AB, Longitudinal fissure. Sy. fis., Sylvian fissure. Sy. h. fis., Horizontal limb of fissure of Sylvius. Sy. a. fis., Ascending limb of fissure of Sylvius.

DE, Perpendicular line from depression in front of external auditory meatus to middle line of top of head. FG, Perpendicular line from posterior end of base of mastoid process to middle line of top of head.

FH, Fissure of Rolando. p. o. fis., Parieto-occipital fissure. + Most prominent part of parietal eminence.

Fissure of Sylvius.—To find this fissure draw a line from a point one inch and a quarter behind the external angular process of the frontal bone, to a point three-quarters of an inch below the most prominent part of the parietal eminence. Measuring from before backwards the first three-quarters of an inch will represent the main fissure, and the rest of the line will indicate the horizontal limb. The ascending limb will start from the posterior end of the line indicating the main fissure—that is, two inches behind and slightly above the external angular process, and will run vertically upwards for about an inch.

Fissure of Rolando.—To find the fissure of Rolando, and at the same time the region of the ascending frontal and parietal convolutions, first, indicate on the surface of the scalp the longitudinal fissure and the horizontal limb of the fissure of Sylvius; next, from the base line draw two perpendicular lines to meet the line for the longitudinal fissure, one (DF, Fig. 3) from the depression in front of the external auditory meatus, and another (FG, Fig. 3) from the posterior border of the mastoid process, at its root. We shall thus have described on the surface of the head a four-sided figure, bounded above and below by the lines for the longitudinal fissure and horizontal limb of the fissure of Sylvius respectively, and in front and behind by the two perpendicular lines. If we now draw a diagonal line (FH, Fig. 3) from the posterior superior angle to the anterior inferior angle of the space, this diagonal line will lie over the fissure of Rolando. In the majority of cases the fissure of Rolando does not actually run into the fissure of Sylvius, a convolution bridging across at that point; hence the lowest half-inch or so of the line would cross this convolution. In those cases where the two fissures join, the fissure of Rolando would be indicated by the whole length of the line.

External parieto-occipital fissure.—Reid says:—"This fissure is more variable in its position than any of the other principal fissures of the cerebrum, and consequently indication of it on the surface of the scalp must be only approximately accurate. If we continue the line for the horizontal limb of the fissure of Sylvius onwards to the line indicating the longitudinal fissure, we shall find that on trephining over the inner inch of this line we shall expose the external parieto-occipital fissure, or some part of it (p. o. f., Fig. 3). In most cases we have seen the whole of the fissure in the opening thus made, in others it showed itself in the posterior half of the opening, and in still fewer cases it was seen in the anterior half."

The position of the principal convolutions (and therefore of the secondary sulci) of the several cerebral lobes may now be determined with comparative ease.

A line drawn from the supra-orbital notch (situated at the junction of the inner with the middle third of the orbital margin) backwards to within three-quarters of an inch of the line for the fissure of Rolando, and parallel with the line for the longitudinal fissure, will indicate the first frontal fissure. The frontal part of the temporal ridge will indicate the second frontal fissure. The first, second or third frontal convolutions will thus be mapped out. The ascending frontal convolutions will occupy a space about three-quarters of an inch broad parallel with, and in front of, the line for the fissure of Rolando (Fig. 4).

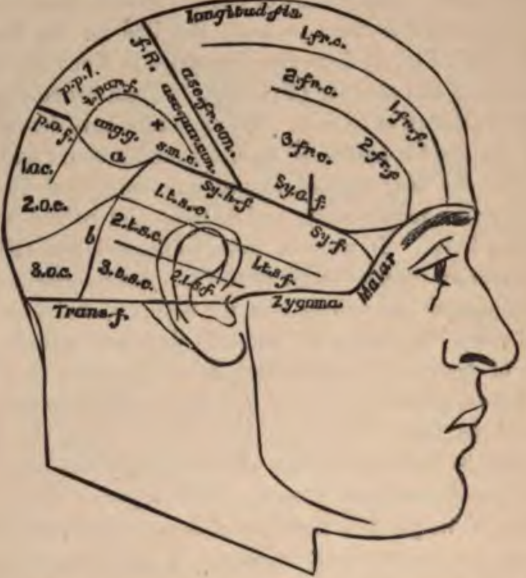

FIG. 4. RELATIVE POSITION OF FISSURES AND CONVOLUTIONS OF THE OUTER SURFACE OF THE RIGHT HEMISPHERE [after Reid].

+ Most prominent part of parietal eminence. a, Convex line bounding parietal lobe below. b, Convex line bounding temporo-sphenoidal lobe behind. 1fr. c., First frontal convolution. 1fr. f., First frontal fissure. f.R., Fissure of Rolando. Sy. f., Sylvian fissure. Sy. h. f., Horizontal limb of Sylvian fissure. Sy. a. f., Ascending limb of Sylvian fissure. p. o. f., Parieto-occipital fissure. i. par. f., Intra-parietal fissure. ang. g., Angular gyrus. s. m. c., Supra marginal convolution. 1 t. s. c., First temporo-sphenoidal convolution. 1 t. s. f., First temporo-sphenoidal fissure. 1 o. c., First occipital convolution. p. p. l., Postero-parietal lobule.

The boundaries of the frontal lobe are obvious; it extends backwards as far as the fissure of Rolando, and downwards as low as the fissure of Sylvius. The parietal lobe is of a V-like shape, being bounded in front by the fissure of Rolando, below by the posterior half of the Sylvian fissure and the parieto-occipital fissure, whilst in the intermediate region (represented in Fig. 4 by the curved line *a*) its boundary is subject to much variation in different brains. This region is occupied by the junction of the postero-parietal lobule with the first annectent convolution of the occipital lobe, and by the junction of the angular gyrus with the second annectent convolution.

The arrangement of these convolutions is extremely variable with regard to one another—"the angular gyrus usually projecting somewhat more posteriorly than the postero-parietal lobule."

The situation and direction of the *intra-parietal fissure* is shown in Fig. 4 (i. par. f.) and needs no verbal description. It is parallel in its anterior third, or thereabouts, with the fissure of Rolando, and about three-fourths of an inch behind it. "In the space above the intra-parietal sulcus we shall have, in front, parallel with the whole length of the fissure of Rolando, the ascending parietal convolution, and behind the postero-parietal lobule. The space below the sulcus will indicate, in its anterior part, the supramarginal convolution (s. m. c.), filling up the most prominent part of the parietal eminence, and, in its posterior part, the angular gyrus (ang. g.)."

The temporo-sphenoidal lobe (or as it is now for the sake of brevity commonly called, the temporal lobe) is, like the parietal, "somewhat difficult to indicate posteriorly, because it becomes continuous there with the parietal and occipital lobes without any distinct line of demarcation." "It will be bounded above by the line

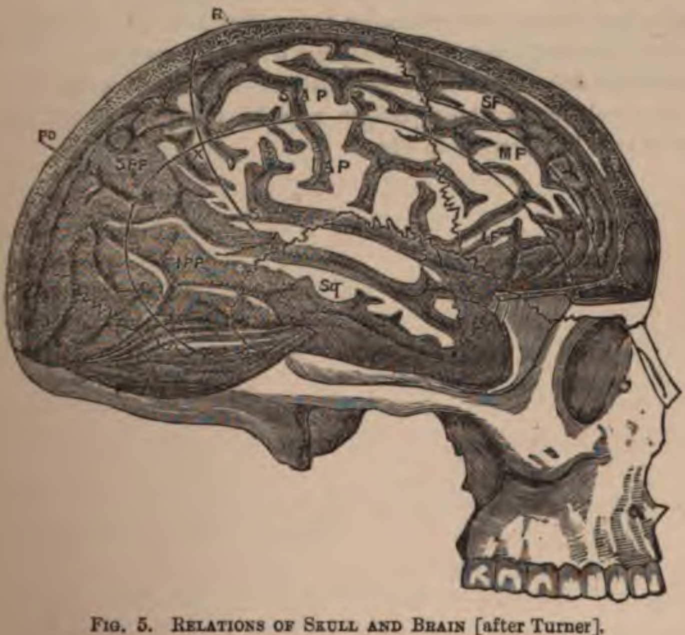

FIG. 5. RELATIONS OF SKULL AND BRAIN [after Turner].

of the main trunk and horizontal limb of the fissure of Sylvius, below by the upper border of the zygoma and a line carried back from the posterior end of that to a point midway between the external occipital protuberance and the posterior border of the mastoid process at its root. The anterior border or apex of the lobe will extend as far forwards as the posterior superior border of the malar bone. Behind, the lobe will be bounded by a slightly convex line (b, Fig. 4) with the convexity directed backwards, extending from the posterior end of the Sylvian line to the posterior end of the line indicating the lower boundary of the lobe. A line running about one inch below and parallel with the line for the main trunk and horizontal ramus of the fissure of Sylvius will indicate the first temporal fissure (1t. s. f.), and another line about three-quarters of an inch below and parallel with the last will indicate the second temporal fissure (2t. s. f.). Thus the first second and third temporal convolutions are mapped out."

The occipital lobe will occupy the remaining surface of the scalp. It will be bounded above by the parietal lobe, below by the superior curved line in its inner half—that is, the part of the line unoccupied by the lower limit of the temporal lobe. Internally the line of the longitudinal fissure, and externally the convex line, above described, for the posterior border of the temporal lobe will bound it. Within the occipital lobe the first, second and third occipital convolutions, are the only parts whose position need be referred to, and these may be seen by reference to Fig. 4.

PATHOLOGICAL DIAGNOSIS.

GENERAL CONSIDERATIONS CONCERNING THE SEVERAL CONDITIONS WHICH CAUSE PARALYSES OF ENCEPHALIC ORIGIN.

We enumerate below the several pathological causes of Encephalic Paralysis.

Apart from traumatic causes, they are set down, and will be considered, in the proximate order of their frequency.

I.—Traumatic Causes (including under this head Gunshot and Sabre Injuries, together with the effects of blows and falls of different kinds).

II.—Ruptures of Bloodvessels, either on the surface or within the substance of the Brain (Meningeal and Intra-Encephalic Hæmorrhages).

III.—Occlusions of Bloodvessels, either by Thrombosis or by Embolism (Softening).

IV.—Tumours of the Brain and its Meninges.

V.—Abscesses within the Cranium.

VI.—Meningo-Encephalitis.

VII.—Disseminated Sclerosis.

VIII.—Dilatations of Bloodvessels (Aneurysms).

IX.—Hydatid Cysts.

X.—Cysticerci.

XI.—Simple Cysts.

XII.—Congenital or early Infantile Pathological States of the Brain.

The above classes will be found to include all the organic causes of paralyses of encephalic origin.

After the effects produced by such injuries or pathological conditions in the several parts of the encephalon have been considered we shall then be in a position to consider with more advantage the forms of paralysis which are supposed to be producible by mere functional perturbations—those known, for instance, under the names of Hysterical Paralysis and Reflex Paralysis. Clearly we must know all the different forms of paralysis due to, or producible by, definite lesions, before we can safely attempt to say that this or that form is caused by functional perturbations only.

Under the first three classes are included all the cases in which the paralysis is abrupt in its mode of onset, as an almost invariable rule. The paralyses pertaining to class VIII often do not show themselves at all until the time arrives when the aneurysm ruptures, and death is imminent. The forms of paralysis associated with the other classes have all of them usually a more or less gradual onset.

The common causes of Paralyses of Encephalic Origin may, therefore, be ranged into two classes from the point of view of the rapidity or slowness of their development.

Onset of Paralysis Rapid.	**Onset of Paralysis Gradual.**
Traumatisms.	Abscesses and Chronic Meningitis.
Hæmorrhages.	Tumours.
Embolisms.	Hydatids and Cysticerci.
Thromboses.	Disseminated Sclerosis.

I. TRAUMATISMS.

In this class of cases the widest possible amount of variation exists, according as we may have to do with gun-shot injuries, with sabre wounds, with the effects of blows or falls upon the head, of different degrees of severity. Each case must therefore be the subject of careful study as it presents itself. In any of them we may have to do with fractures of the skull, with lacerations of brain tissue, and with hæmorrhages upon or into its substance, as immediate results, and some of these lesions may give rise to severe forms of paralysis. The secondary results of such injuries are also of a most formidable character. An immediate consequence may be inflammation either of the injured brain substance (Cerebritis) or of some portions of the meninges (Meningitis), or of both together (Meningo-Cerebritis).

A more remote result may be the formation of an Abscess in some portion of the brain substance. The degree of remoteness of this possible sequence varies much in different cases. It has been known to form within seven days of the original injury; in other cases not till the lapse of several years. These are extremes in both directions; the commonest period within which an abscess manifests itself after an injury is from 6-12 weeks.

Other more remote effects of blows or falls upon the head which may not begin to manifest themselves, perhaps, for one, two, three or more years (though in exceptional cases it may be earlier) are Tumours of different kinds and Disseminated Sclerosis. It is now commonly admitted by many good authorities that tumours of the meninges or of the brain substance may in certain cases be initiated in this manner. It is not a matter of common knowledge or admission that Disseminated Sclerosis may occasionally have such an origin, yet this is a view which has been strongly impressed upon the writer from his own experience. Antecedent severe blows or falls count for something, therefore, in arriving at a diagnosis of either of these formidable diseases.

II. RUPTURES OF BLOODVESSELS, PRODUCING HÆMORRHAGES.

Hæmorrhage into or upon the surface of the brain varies most widely both in situation and in amount in different cases. There are nevertheless particular regions in which it is more especially prone to occur. Even where giving rise to obvious symptoms the blood effused may vary in amount from a minute clot not larger than a pea occurring in the pons Varolii, to an effusion of many ounces of blood which tears up the substance of one hemisphere and subsequently fills the lateral ventricles. Intra-cranial effusions of blood may also be single or multiple in the same individual.

The intra-cranial hæmorrhages that we have to consider are divisible into two distinct categories, which for various reasons require to be carefully discriminated from one another. They are 1. Meningeal Hæmorrhages, and 2. Intra-Cerebral or Intra-Cerebellar Hæmorrhages. Each of these classes requires some separate consideration.

1. Meningeal Hæmorrhages. These are due either to rupture of a branch or branches of one of the meningeal arteries, or else to the giving away of some venous or arterial trunk or branch in the pia mater. At other times a meningeal hæmorrhage is due to the bursting of an aneurysm situated on one of the larger vessels in or near the circle of Willis.

Meningeal hæmorrhages tend to occur at a decidedly earlier age than cerebral or cerebellar hæmorrhages. Even in very young children whose vessels are healthy, bleeding into the arachnoid may occur from any unusual amount of strain. This takes place occasionally at the time of birth, especially during the mechanical congestion often inseparable from prolonged labours. According to Cruveilhier arachnoid hæmorrhage is the cause of death in about one-third of those infants who die almost immediately after birth. A little later in life a similar accident may occur during paroxysms of whooping-cough or during other spasmodic states in which the return of venous blood from the head is impeded. In older children an arachnoid hæmorrhage not unfrequently follows falls on or blows about the head; or it may result from the rupture of aneurysms on some one or other of the large vessels at or near the base of the brain. This latter event rarely takes place in individuals under fourteen years of age, though from this period onwards it is apt to occur pretty equally at different ages.

Such aneurysms are decidedly more common in males than in females; and in respect to other causes of meningeal hæmorrhage the disproportion in the same direction is still greater. Thus of 163 recorded cases of meningeal hæmorrhage collected and analysed by Gintrac, nearly three fourths were males. If we add to these cases the other 77 instances of hæmorrhage upon the surface of the brain analysed by the same author—viz., those produced by rupture of aneurysms on the larger cerebral vessels—we get a total of 240 cases of meningeal hæmorrhage, which were distributed in respect to age in the manner shown in the subjoined table (see p. 27).

A large arachnoid hæmorrhage is capable of producing sudden coma with unilateral or bilateral paralysis, and may be followed by speedy death.

2. Cerebral and Cerebellar Hæmorrhages. Bleeding takes place into the substance of the brain with great frequency as compared with the frequency of such an occurrence in other organs. This is doubtless due partly to the softness of the brain and partly to the fact that the small arteries lie almost unsupported within perivascular canals of a larger diameter, which ramify with them throughout the brain substance. Within these canals the only immediate support to the small vessels must be derived from the pressure of the cerebro-spinal fluid by which they are surrounded.

The canals that ramify through the substance of the corpora striata are frequently three times as wide as the vessels which they contain, as I have found by actual measurements. Such an anatomical arrangement peculiarly favours the production of the small miliary aneurysms which have been shown by Bouchard, Charcot and others to be so frequent in the cases of cerebral hæmorrhage occurring in elderly people. These miliary aneurysms vary in size from a small pin-head to that of a mustard seed. They may occur in almost any part of the brain and at times they exist in considerable numbers. Where they exist the rupture of one or other of them may lead to hæmorrhage into the brain at any time. The production of these vascular dilatations is especially favoured by the previous occurrence of fibroid changes in the walls of the vessels. Such changes however, as well as fatty or atheromatous degenerations, which are also very frequent in the cerebral vessels, tend to increase in direct proportion to the age of the individual.

The facts above stated would seem to render it possible that cerebral hæmorrhages should occur at almost any age, looking to the softness of the brain and the further lack of support to its vessels of which I have spoken. We might also expect that these influences would become more and more operative with advancing age, owing to the progressive increase of vascular degeneration of the fibroid and the fatty type which this so commonly entails. Judging from the elaborate statistics published by Gintrac this seems to be what really occurs. He has published and analysed the records of 658 cases of hæmorrhage into different parts of the brain, and he found them to be thus distributed according to age. For the sake of comparison we give here, side by side, the table supplied by the same authority concerning meningeal hæmorrhages.

MENINGEAL HÆMORRHAGES.

Birth	to 10 years		10	cases.
11	years to 20	„	19	„
21	„ „ 30	„	27	„
31	„ „ 40	„	52	„
41	„ „ 50	„	33	„
51	„ „ 60	„	41	„
61	„ „ 70	„	32	„
71	„ „ 80	„	26	„
			240	

HÆMORRHAGES INTO THE BRAIN.

Birth	to 10 years		15	cases.
11	years to 20	„	24	„
21	„ „ 30	„	44	„
31	„ „ 40	„	74	„
41	„ „ 50	„	98	„
51	„ „ 60	„	129	„
61	„ „ 70	„	152	„
71	„ „ 80	„	110	„
81	„ „ 90	„	12	„
			658	

Childhood, youth, or early adult age does not, therefore, by any means preclude the diagnosis of cerebral hæmorrhage in any given case, though reference to the figures above quoted will show that at these periods of life meningeal hæmorrhages are proportionately much more frequent.

On the other hand, as we approach middle life and old age, hæmorrhages into the substance of the brain becomes more and more frequent. The diminution in numbers after the 70th year is only apparent, owing to the much smaller number of people who live after this age has been attained.

With regard to the most common seats of hæmorrhage within the skull, it is not easy to arrive at correct information because we are apt to find an undue proportion of rare cases put upon record. Still if we look to large numbers we obtain data of a certain value. In a total of 751 cases of hæmorrhage within the cranium, Gintrac found it recurring in different situations in the following manner—Meninges, 172; cortical substance of the hemispheres, 45; middle lobes of the brain, 127; posterior lobes, 33; anterior lobes, 17; corpora striata, 72; optic thalami, 38; pons and cerebral peduncles, 76; medulla, 2; cerebellum, 55.

The great frequency here indicated of hæmorrhage into the 'middle lobes' of the brain is a very striking fact. But it is in part explicable by the far greater bulk of the middle portion of the brain—roughly spoken of as the middle lobe—as compared with that of the anterior or the posterior segments of the hemisphere; though it is still further explicable by reference to the mode of distribution of the vessels to these different parts, the description of which will be found further on. The latter consideration also suffices to explain the greater frequency with which the posterior lobes are the seat of hæmorrhage as compared with the anterior lobes.

The frequency of the occurrence of hæmorrhage in the pons and cerebral peduncles is also striking, especially when compared with its extreme rarity in the medulla oblongata. Hæmorrhages in the latter situation are always minute, rarely attaining the size of a pea. In these respects the medulla oblongata is rather allied to the spinal cord than to the brain.

Details as to the distribution of the blood vessels in different parts of the brain will not be considered now. This is a matter of considerable importance, but it will be found to bear principally upon the problem of Regional Diagnosis. The subject will, therefore, be considered as one of the preliminaries to these sections.

III. OCCLUSIONS OF VESSELS, BY THROMBOSIS OR BY EMBOLISM.

The next common cause of paralyses of encephalic origin is occlusion of Vessels, whether this is brought about by Thrombosis or by Embolism. In each case the pathological consequences are much the same whether we have to do with occlusion by the one or the other process. The result is some amount of 'Softening of the Brain,' so long as the occlusion occurs in one of the vessels situated on the distal side of the circle of Willis.

The portion of brain tissue previously nourished by the occluded vessel is cut off from its blood supply; the peripheral portions of this vascular area become hyperæmic and serum is effused, whilst the central portions almost at once begin to undergo fatty and degenerative changes, resulting in the course of a few days in the production of a focus of 'softening' or cerebral 'ramollissement.'

Occlusion of one or other of its large vessels seems to be by far the most common cause of Softening of the Brain, though in some instances, more especially in old people, in whom there are widespread atheromatous changes in the smaller cerebral vessels, a patch or patches of degenerative softening may be found where no occlusion of larger vessels can be detected.

Where Softening is of inflammatory origin, it is almost invariably secondary to other changes, such as traumatisms, adventitious products, or blood clots.

Patches of softening may exist singly, or several patches may be found coexisting in the same brain. The foci themselves may be either very minute, or so extensive as to involve nearly the whole of one hemisphere with, perhaps, part of the other.

It is important to remember that in cases of occlusion of vessels it is the cutting off of the blood supply from a certain portion of the brain tissue—and consequently the functional inertness of this particular brain-region—which gives rise to the paralytic symptoms. Softening is only one of the after effects of the series of pathological changes resulting from this deprivation of blood. It is important to bear this in mind because, should death occur within forty-eight hours from the onset of paralytic symptoms, no appreciable softening may be discoverable, simply because there has not been sufficient time for this pathological condition to have become established.

A few words must now be said concerning each of the common causes of softening of the brain.

Thrombosis is a process which may occur either in arteries, in veins, or in cerebral sinuses, but as a cause of hemiplegia we shall in the large majority of cases have to do with the process as it occurs in one or other of the large cerebral arteries, or else in several of them simultaneously. Occasionally, however, ill-defined forms of paralysis are produced by thrombosis in the longitudinal sinus, extending into the cerebral veins over the vertex, on one or both sides.

The local coagulation or separation of fibrine in certain vessels, of which the process of thrombosis consists, is determined in part by (1) general causes, tending to diminish the force and frequency of the heart's action, or to produce certain alterations in the quality of the blood; and in part by (2) local degenerative changes in the coats of the vessels in which the clotting occurs, or changes of an inflammatory type (endarteritis)—which are probably much more frequent than is commonly supposed, especially in tuberculosis and syphilis.

The general causes come into operation principally in certain cachectic conditions of the system, in which, whilst the heart is weak, the blood is more than usually prone to coagulate. This seems to occur occasionally in the period of convalescence from acute diseases (such as pneumonia or typhoid fever more especially), and also in some delicate women during the first two or three weeks after childbirth. The process is always favoured in either of these conditions by the existence of a slow, irregular, and weak action of the heart.

The chief local causes of thrombosis are pathological changes in the coats of the vessels, whereby their inner wall is roughened, or by which the calibre of the vessel is narrowed. Thus it is favoured by atheromatous or calcareous changes, which roughen the vessel's surface, or expose the blood to the coagulating influence of not-living materials in the vascular wall; as well as by all fibroid thickenings and indurations of vessel walls—whether syphilitic or non-syphilitic. Almost similar results may be entailed by a process of endarteritis apt to occur in syphilis, tuberculosis, and other states of the system.

Whenever it happens that general determining conditions are reinforced by the existence of local degenerative changes, thrombosis is of course most prone to occur; but in some cases general conditions alone seem to suffice without the determining influence of marked degenerative change, and this is the reason why thrombosis is almost as common in early adult life as it is at later periods.

This process may occur in any of the vessels at the base of the brain, or in several of them at the same time—as we not unfrequently find when a thrombus forms in one of the internal carotid arteries. The clot may extend from this trunk not only into the middle and anterior cerebral arteries, but perhaps also into the posterior communicating and the posterior cerebral artery of the same side.

Thrombosis in the Cerebral Sinuses. Thrombosis occurs principally in three of the cerebral sinuses, viz., in the longitudinal, and in one or other of the two lateral sinuses. A similar process is also often set up in tributary veins either as a secondary or as a primary event.

(1.) The formation of a thrombus in the longitudinal sinus usually proves to be a primary phenomenon dependent in the main upon the operation of general causes, such as some alteration in the quality of the blood, combined with slow, feeble, and irregular action of the heart. The operation of these causes has however been known to be favoured in certain cases by local conditions, such as the great development of Pacchionian bodies and their projection into the sinus—an event most likely to be met with, when it occurs at all, in persons past middle life. Thrombosis of the longitudinal sinus may, however, be met with also in the earlier stages of life. The original thrombus not unfrequently prolongs itself through the straight sinus to the torcular Herophili, and thence on either side into the lateral sinuses. In this latter class of cases ventricular effusions of serum (owing to impeded return of blood from the central parts of the brain) and superficial bilateral softenings are especially apt to be met with—the latter occurring principally in those cases in which the thrombus has extended laterally from the longitudinal sinus into the great veins of the cerebral hemispheres which open into it on each side. These softenings are of a peculiar and characteristic kind, consisting generally of a number of small red patches principally confined to the grey matter of the upper parts of the cerebral hemispheres. Occasionally softening of a portion of brain of considerable extent has been produced. Besides the ventricular effusion there may also be an excess of serum beneath the arachnoid, or more rarely small effusions of blood in this situation, together with minute patches of hæmorrhage within the subjacent grey matter. The actual combination of conditions met with, will depend upon the seat and extent of the obstruction, the rapidity with which it has been brought about, and the existence or not of marked weakness in the walls of the veins and capillaries pertaining to the affected territory.

The variation in the symptomatology of this affection in different cases is therefore extreme; the symptoms are sometimes excessively grave, and at other times they have been almost *nil*. Strange as it may seem, coming from such a good observer, Gee says :—" I have known a decolorized softening thrombus to occupy the whole bore of the upper longitudinal sinus, to be attended by large subarachnoid hæmorrhages, and to have caused no symptoms during life."

(2.) Fully as frequent as the event above referred to is the formation of a thrombus in one or other of the lateral sinuses. Here however the process is almost invariably secondary to inflammation of the scalp or cranial bones, whether induced by disease or by traumatic conditions.

Caries of the cranial bones is the principal predisposing cause; moreover in no less than three-fourths of the recorded cases, the temporal bone has been the part affected with caries and that as a result of chronic suppurative disease of the middle ear. In these cases there is often evidence of a more or less circumscribed inflammation of the meninges in association with the chronic disease of the bone. Cerebral softenings and arachnoid extravasations of blood, as a consequence of the thrombosis in the lateral sinus, are however here rarely or ever met with. According to Von Dusch this is explicable by the fact that in these cases the thrombosis starts from the veins in communication with the inflamed spot, and reaches the lateral sinus only after a collateral circulation has had time to establish itself, instead of commencing in the sinus itself, as in the cases previously referred to in which the longitudinal sinus is affected. Both lateral sinuses are rarely affected at the same time in this secondary manner, though this may be the case when a primary longitudinal thrombosis extends into them.

Out of 74 instances of Thrombosis in the Cerebral Sinuses which have been recorded by Lanceraux and other observers no less than 39 are found to have followed in the wake of blows on the head or inflammatory conditions of the scalp and cranial bones. Among these, in 30 cases there was caries of the bones of the skull, in 24 of which it was the temporal bone that was affected as a result of internal otitis.

Embolism may be encountered in any of the vessels beyond the circle of Willis, though it is especially prone to occur in the middle cerebral (Sylvian) arteries. It is met with more frequently in the artery of the left than in that of the right hemisphere, so that hemiplegia from this cause is most frequently right-sided in its distribution. Embolism may take place at any period of life, though it is much more frequent under the fortieth year than after that age.

Emboli themselves vary both as to their nature and origin. They may consist of fibrinous masses, swept away from vegetations on the valves of the heart or from bodies of the same nature on the lining membrane of the auricle, or of débris of any kind from some part of the aorta anterior to the origin of the great vessels that carry blood to the brain. More rarely the embolus consists of a detached fragment of a thrombus formed in one of the pulmonary veins. This enters the left cavities of the heart and may be thence swept onwards into one of the large cerebral vessels.

At other times numerous embolic particles or fragments are derived from the crumbling of a fibrinous concretion which has formed within the left auricle or ventricle. In instances of this latter description, or where a large atheromatous depôt in the aorta discharges a quantity of débris into the circulation we may have multitudes of small embolisms in several vascular areas instead of a stoppage of one of the main arteries by a larger occluding mass. This kind of accident has been met with rather in experiments with lower animals (Prevost and Cotard) than at the bedside. It is well, however, to be aware of such a possibility; and to bear in mind that these cases in which the embolisms are both numerous and wide-spread, are particularly liable to produce a rapidly fatal apoplectic condition.

When it happens that the occlusion of a main vessel is not perfectly brought about by the impacted body, the stoppage of the blood current is apt to be soon completed by the formation of a thrombus on the distal side of the embolus, causing necessarily an aggravation of pre-existing symptoms.

What has been said above concerning the occurrence of ruptures of intra-encephalic blood vessels, and their occlusions by embolism or thrombosis, must make it obvious how important it is, with a view to the proper interpretation of the symptoms of cerebral disease, to study the areas of distribution of, and the degree of inosculation existing between, the several arteries of the cerebrum and the cerebellum. Bearing in mind the fact that embolism and thrombosis produce their functional and pathological effects principally when they occur in 'end' arteries in which a collateral circulation cannot be set up, it becomes clear that something like a detailed knowledge concerning the vascular supply of the brain is essential. Such knowledge will prove useful in explaining the extent and distribution of foci of softening, and the relative frequency with which they implicate different parts of the brain. Light will also, thereby, be thrown upon the causes of the frequency of hæmorrhages in different regions of the brain, and upon the variations met with in the amount of blood apt to be poured out in different situations. It is true these facts bear as much upon the problem of regional diagnosis as they do upon that of pathological diagnosis. For this reason it has been thought best that they should be considered in several preliminary sections anterior to discussing the problems of Regional Diagnosis relating to different brain regions.

IV. TUMOURS OF THE BRAIN AND ITS MENINGES.

During life it is often absolutely impossible to determine whether a new formation imagined to exist within the cranium has originated in the midst of the brain substance or, having commenced in one or other of the meninges, has merely grown into or pressed upon some part of the brain secondarily. In both cases, other things equal, the symptoms produced may be almost identical. Very rarely an extra-cranial morbid growth may extend inwards through the orbit, and by its pressure upon the brain may give rise to symptoms similar to those that would be produced by a growth originating within the cranium and similarly interfering with the anterior lobe of the cerebrum.

Brain tumours exist either as distinct circumscribed growths with sharply defined outlines; or they may be, as it were, infiltrations passing insensibly at their circumference into the surrounding brain tissue. In the former case the new growths are occasionally enclosed in a fibrous envelope of more or less thickness. The brain tissue around a tumour may be healthy; it may be indurated or firmer than natural; or lastly it may be softened to a variable extent. The softening may be simply white or it may exhibit various shades of red or yellow colour, according to the conditions of its origin, its age, and other peculiarities. On the other hand, hæmorrhages are not uncommon into the substance of certain soft and vascular brain tumours.

A few details may now be given concerning the different kinds of new growth met with within the cranium—that is concerning those commonly met with, viz., 1. Tubercular or Scrofulous Tumours; 2. Syphilitic growths; 3. Cancerous growths; 4. Gliomata; 5. Sarcomata; 6. Myxomata; 7. Tumours of the Pituitary Body; 8. Exostoses. The other growths occasionally met with are scarcely worth mentioning from a diagnostic point of view.

1. Tubercular or Scrofulous Tumours. These are much more frequently met with in children than in adults, though they are rarely found in children under two years of age, and have been said to be most common from the third to the seventh year inclusive. Such growths are not usually found in the brain of a child unless they exist in other organs of the body at the same time.

In size the separate masses of cerebral tubercle vary between that of a millet seed and a large hen's egg. The most common sizes range from a large pea to a small walnut. They are mostly rounded but sometimes lobulated in form. They may be found in almost any part of the brain but are more frequent in the cortical grey matter than in other parts. The Cerebellum, also, seems with especial frequency to be the seat of tubercle. Tubercular growths may be single, or two or three masses may coexist; in other cases a large number of small tubercular masses may be found in different parts of the brain.

The growth of these masses takes place by new accretions at the surface of small miliary tubercles, which speedily fuse with the mass already existing, and become like it of an opaque yellow colour. When the period of active growth ceases, these tumours are apt to become surrounded by a dense fibrous envelope which cuts them off from the surrounding brain tissue.

A distinctly phthisical family history is often to be obtained, where intra-cranial tubercular growths exist. Sometimes the mode of death in such cases is by the supervention of tubercular meningitis—in which there is the development of multitudes of minute granulations in connection with the vessels of the pia mater. In other cases tubercular tumours not previously existing may develop within some part of the substance of the brain during an attack of tubercular meningitis, giving rise to the occurrence of paralysis in some part or parts of the body.

2. Syphilitic Growths. These, unlike the last, are rarely met with within the substance of the brain, though on exceptional occasions a gummatous tumour has been met with in the thalamus, in the corpus striatum, in the cerebellum or in some other region—varying in size from a pea to a large filbert.

What we have to do with principally under the present heading, are syphilitic thickenings and overgrowths from the meninges—forms of pachymeningitis, in fact, with gummatous lymph-like matter glueing the dura mater to the arachnoid and pia mater and even invading the cortex of the brain itself for some depth. It seems probable that these gummatous overgrowths often take their origin in the pia mater and arachnoid. They may also be found to involve the outer layers of the dura mater, and extend thence to the inner table of the skull so as to form adhesions with it, or more or less erosion with actual replacement of bone.

Such thickenings and overgrowths as are first referred to, often produce all the signs of a definite tumour within the cranium. Whilst they may occur at almost any part of the circumference of the brain, they are especially common in the parietal region. They are apt also to be found on some part of the under surface of the brain.

These changes are results of acquired syphilis but not of the inherited disease. Age and sex must, therefore, count for much in reference to our diagnosis of this pathological condition. And in the case of females we must have regard to their station in and probable habits of life, since these are conditions that may legitimately enough influence our judgment.

It is generally believed that syphilitic pachymeningitis occurs only after the lapse of several years. Probably six to ten years from the date of the primary affection is the period when it is most apt to show itself. It very rarely occurs within three years of the date of infection.

3. Cancerous Growths. Intra-cranial cancers may take origin in some one or other of the cranial bones, in the dura mater, or within the brain itself.

Where the dura mater is the seat of origin, the growth often develops into the form of a fungus hæmatodes which attacks and perhaps penetrates the bones externally, whilst in its growth inwards it may successively infiltrate and glue together the subjacent arachnoid and pia mater as well as certain portions of the surface of the brain. Almost the same effects would be produced in the case of a cancerous growth springing primarily from the inner table of the cranial bones.

In both cases the new growth may not actually infiltrate the surface of the brain, it may merely press upon and indent its surface. Although there are obvious pathological differences between tumours pertaining to these two categories, still they may not, and probably will not, lead to any definite differences in the grouping of signs and symptoms.

The cancerous growths in the situations mentioned above may be primary, but they are more likely to be secondary to cancer occurring elsewhere. In any case they are decidedly rare. Sometimes the new growths originating in the inner layers of the dura, or in the visceral arachnoid, have the characters of epitheliomata.

Within the brain itself different forms of cancer may occur, though encephaloid is not so rare as the other varieties. The growth may be circumscribed or it may infiltrate the parts of the brain in which it occurs. They are usually solitary though sometimes multiple; they vary much in size and may equal that of an orange or be even larger. Occasionally even the greater part of one hemisphere may be implicated. Almost any part of the brain may be involved, but the cancerous tumours found in the substance of the cerebral hemispheres are generally the largest. Those which occur in such regions as the pons and medulla are usually comparatively small, because death is much more rapid when such parts are involved.

Effusions of blood may be met with in the midst of soft cancerous growths, and not unfrequently cysts become developed in their interior which are found to contain a thick glairy fluid. The surrounding brain tissue may be natural, or it may be more or less softened. The occurrence of such hæmorrhage, or the supervention of such softening, but especially the former, may be associated with an aggravation of pre-existing paralytic symptoms.

Cancerous growths in the brain, whilst far from common, are, according to Lebert, decidedly most frequent in the second half of life, though they are met with occasionally in youth or even in childhood. Walshe, also, found that out of 56 persons affected with cerebral cancer, 28 died between the ages of 40 and 60 inclusive; while 5 died before the 10th year, and 5 between the 10th and 20th years. In about one half of the total number of cases cancer of the brain is primary.

The duration of life varies considerably; thus in 6 out of 11 cases inquired into by Lebert the growth seemed to have proved fatal in about 6 months, while in 4 the symptoms extended over a period of from 2 to 5 years.

These facts in regard to age at time of onset, and also in regard to the possibly slow progress of the disease, are especially worthy of being borne in mind in regard to cancer of the brain, as also is the fact that the growth is often primary.

4. Gliomata. These tumours are confined to the brain substance. They seem, indeed, to be overgrowths from the neuroglia or connective tissue of the brain. They are seldom sharply defined, but almost invariably infiltrate the surrounding brain tissue—from which they often do not differ notably in appearance. They are usually solitary and of slow growth, so that they may exist for some time without producing any very appreciable symptoms. Gliomata may attain the size of an orange, or they may be even larger than this. They are often met with in some part of the cerebral hemispheres, and not unfrequently in one or other of the posterior lobes. At other times they may be found in one of the basal ganglia, in the pons, or in the cerebellum; but they are less common in the medulla oblongata.

There are two principal varieties of glioma, the soft and the hard.

The soft variety is the commoner of the two, and there is distinct evidence in favour of the view expressed by Virchow to the effect that it is prone to arise as a result of local causes, produced by injuries. The writer believes that it may occasionally take its origin in the sites of previous minute lacerations of brain substance, starting from cicatricial overgrowths of neuroglia.

The hard variety contains a variable quantity of fibres, and the specimens in which these are most abundant approximate in character to fibromata. Virchow believes that this form may also have an inflammatory or sub-inflammatory origin. Hæmorrhages are very apt to occur into the substance of these growths, especially in the softer forms.

Gliomas may occur almost at any age, but seem to be decidedly more common in the first than they are in the second half of life. They may even be congenital, and they are, after scrofulous growths, decidedly the commonest form of tumour occurring in the brain of children and young adults.

5. Sarcomata. The sarcomata like the tumours above referred to are also divisible into soft and hard varieties. The soft varieties have structural alliances with gliomas, while the harder varieties are similarly closely related to fibromata.

Sarcomas take origin both from the meninges and from the brain substance, but they more frequently commence in the former than in the latter.

Sarcomas of the dura mater are commonest in one of the middle fossæ at the base of the skull. They form more or less rounded tumours springing from the inner layers of this membrane, mostly varying in size from a small hazel-nut to that of a large walnut. They press upon and indent the brain substance, and when situated at the base they are also especially apt to press upon one or more of the cranial nerves.

Of the sarcomas which originate in the substance of the brain some form circumscribed easily separable tumours—and these are usually of the hard variety. Others infiltrate the brain substance after the fashion of the gliomas—and these are commonly soft sarcomas. Both kinds are furnished with large and numerous thin-walled vessels, and hæmorrhages are very apt to take place into the substance of such growths. This is not mentioned as a mere matter of pathological interest, but rather because on the clinical side it may show itself by some abrupt aggravation of pre-existing symptoms, owing to a sudden increase of pressure within the cranium. This aggravation of symptoms may be very distinct in certain cases where such increase of pressure tells, as it does sometimes, upon important parts of the brain.

Sarcomata may appear in almost any part of the cerebral hemispheres or cerebellum. They have been found infiltrating the optic thalamus, the corpus striatum, the pons, and other important regions.

Sometimes the sarcomas met with in the brain are deeply pigmented (so called "melanotic tumours"); such growths are prone to be multiple, and to co-exist moreover with similar growths in different parts of the body. This latter peculiarity is, however, common to all the varieties of sarcoma that occur within the cranium.

Sarcomas like gliomas are apt to undergo a sort of mucoid degeneration, and later on, where such changes have been carried to an extreme degree, there may be the development of a kind of pseudocyst with fluid contents.

Sarcomas occur at all ages, and are by no means unfrequent in quite young children.

6. Myxomata. Within the skull myxomata are rare growths. Still they are occasionally found in connection with the inner layers of the dura mater, while at other times they take their origin in some part of the brain substance. Myxomata are sometimes closely related to soft gliomata in structure.

It is important to remember that they are distinctly rare growths, and that they carry with them no peculiar features of any kind; so that it would be almost impossible in any given case to pass beyond the general diagnosis of tumor cerebri and to say that the tumour was probably a myxoma.

7. Tumours of the Pituitary Body. The pituitary body is liable to become hypertrophied or the seat of some new formation by which it assumes the appearance of a tumour, having the size of a walnut, or even of a hen's egg. Tumours in this region will sometimes extend back as far as the pons and laterally to the commencement of the Sylvian fissures, so as to occupy the whole interpeduncular space. Growths of such a size besides giving rise to the general signs indicative of tumor cerebri may press upon one optic nerve as well as upon some of the motor nerves of the same eyeball, and this combination of signs with perhaps mere paresis of the limbs on the opposite side should suggest the possibility of a tumour in the pituitary body causing pressure more on one side than on the other. A somewhat similar condition might be induced (so far as pressure upon nerves is concerned) by a carotid aneurysm within the cavernous sinus.

8. Exostoses. Exostoses from the interior of the skull large enough to press severely upon the brain and act like intra-cranial tumours are decidedly rare. Still they sometimes attain large dimensions. They may be single or multiple, and may coexist with similar overgrowths from the external table of the skull. They have been known to occur in the site of some previous severe blow, and to go on slowly increasing in size for ten, fifteen, or more years. It is important to remember that such a growth may develop internally as well as externally.

Tumours of the Pineal body are mostly small and rarely give rise to any definite symptoms.

Fibromata are extremely rare within the cranial cavity; though tumours closely approximating to this type are met with in the form of hard sarcomas and hard gliomas respectively.

The other new-growths which have from time to time been encountered within the cranial cavity are all small; many of them are of extreme rarity, whilst those that are common are almost, if not wholly, devoid of clinical significance. To the former category belong Psammomata, Lipomata, Papillomata; and to the latter Osteomata and Pacchionian bodies.

V. ABSCESSES WITHIN THE CRANIUM.

Abscesses of the brain are what we have to do with principally under this head; still in other cases the collection of pus may be more or less circumscribed either between the dura mater and the brain, or somewhere within the arachnoid cavity.

Abscess within the cranium is a comparatively rare disease, and when it occurs, in the large majority of cases, it follows in the wake of traumatisms, of chronic disease of the middle ear, or of pyæmia. In the small minority of cases the abscess occurs without definite assignable cause or antecedent condition—these are the so-called 'idiopathic' abscesses.

In pyæmia the abscesses are usually small and multiple, often existing near the surface, and varying in size from a mustard seed to a small hazel-nut. Here the general condition is the dominating clinical feature. The amount of head symptoms vary much in different cases of pyæmia and in some of those in which they are well marked minute cerebral abscesses may be found. That is about all that can be said concerning the diagnosis of such multiple abscesses.

Where chronic middle-ear disease has existed, or where abscesses occur after traumatisms of different kinds, they are usually single. This is also the case where we have to do with what may appear to be an idiopathic abscess of the brain—these latter cases, indeed, may probably in most instances be capable of being referred to one or other of the previous categories. Single abscesses of the brain may be situated in almost any part of the cerebral hemispheres or in the cerebellum. They are rare in the medulla or pons, and also rather rare in the thalamus and the corpus striatum. They necessarily vary much in size, but they may attain the dimensions of a small orange or be even larger. Rarely an abscess may burst into the lateral ventricle or on to the surface of the brain. In the early stages of their formation the brain tissue around may present signs of inflammation especially in the traumatic cases. Later on, the abscess may become more or less distinctly encysted, or there may be some amount of softening of the brain tissue around it.

Abscess of the brain of traumatic origin may occur under the following conditions. They may follow upon a fracture of the skull with or without depressed bone; in these cases the abscess may be near the seat of injury and develop pretty quickly, often as a result of some laceration with subsequent inflammation of brain substance; or it may occur in a region remote from the original injury and develop sometimes after a long interval of weeks or months. Just the same history as that last mentioned may occur also in other cases of severe blows on the head in which, however, no fracture of the skull exists. An abscess of the brain may also follow a penetrating wound of any kind.

Chronic suppurative disease of the middle ear (perhaps following scarlet fever, measles, or small-pox) may at any time be lighted up into fresh activity by some blow, exposure to cold, or state of lowered general health. At other times in patients suffering from such chronic inflammation of the tympanic cavity, disease of the petrous portion of the temporal bone or of the mastoid cells may be insidiously extending, and at last cerebral symptoms may become developed—one of the causes of such symptoms being the development of an abscess either in the cerebrum or the cerebellum. According to Toynbee, extension of the disease to the brain rarely occurs so long as there is a free vent for the discharge from the tympanic cavity. Where disease exists in the mastoid cells and such extension occurs, according to the same authority, the cerebrum is most prone to be the seat of the abscess in early life and the cerebellum during later periods of life.

Suppuration in almost any part of the body may occasionally conduce to the development of one or more abscesses in the brain, and that even when there is no development of a general pyæmic condition. Still in these cases we probably have to do with an abortive form of the pyæmic process.

An abscess of the brain resulting from injury may form even at the expiration of a week; the commonest time for it to reveal itself is, however, in 6-12 weeks. On the other hand, in rare cases, it seems only to manifest itself in two or even three years after the patient has received some blow upon the head or concussion of the brain.

VI. MENINGO-ENCEPHALITIS.

Localised Meningo-Encephalitis is due in the main to three causes. It occurs (1) as a syphilitic pachymeningitis, (2) as a result of traumatisms (without or with depression of bone), and (3) in association with tubercular meningitis.

The most definite symptoms are produced in all these cases where the Meningo-Encephalitis involves some part of the excitable area in the fronto-parietal region of the cortex (Laudouzy, 'Convulsions et Paralysies liées aux Méningo-Encéphalites,' 1876). Elsewhere it may be associated with no very marked symptoms.

To the first variety reference has already been made (p. 36); concerning the second nothing special need be said; whilst in regard to the third, we have in these cases to do with the signs of the general malady, plus certain extensions of the meningitis beyond the basal region, giving rise to localised convulsions, or paralyses which (as when due to other cortical lesions) are apt to be incomplete, partial and variable.

VII. DISSEMINATED SCLEROSIS.

Disseminated or insular sclerosis is now known to be a far from rare cause of paralysis of encephalic origin. The patches of sclerosis met with in the Encephalon vary in size from a mustard seed to a focus an inch or more in diameter. They may be found distributed through almost all parts of the brain; but they occur with especial frequency in the bulb and the pons Varolii. For a period these foci of sclerosis may be limited to the brain; but after a time there is always a tendency to implicate the spinal cord—thus giving rise to 'cerebro-spinal sclerosis.' In other cases this same duplex malady becomes established in a different order—the primary affection being that of the spinal cord.

This disease may occur even in early childhood; it is common in adolescents and young adults; whilst it becomes decidedly rare after about the fortieth year. It is rather more common in females than in males. Sometimes it seems to follow in the wake of exposure to wet and cold (even from sleeping in a damp bed); in others it appears to develop after severe mental trouble combined with worry; or after convalescence from some one or other of the acute specific diseases. In some cases neither of such preliminary conditions can be traced.

The diagnosis is often not difficult. It depends in part upon evidence supplied by the regional diagnosis, showing an implication of the pons or of the bulb, and this too by some lesion, capable of dissecting out closely contiguous parts, such as the nuclei or roots of adjacent nerves in the bulb, in a way which a tumour is scarcely likely to do. At the same time we usually find some of the most characteristic signs of tumour absent, viz., severe headaches and optic neuritis.

In what has just been said reference is made to the disease in its purely cerebral form. Where the spinal cord is affected with insular sclerosis as well as the brain, the diagnosis is decidedly easier of recognition, from the mere fact that we have to do with a cerebro-spinal affection of a chronic type—and that the number of such affections is so very limited.

VIII. DILATATIONS OF VESSELS (ANEURYSMS).

Intra-cranial aneurysms are of two kinds. There are first the miliary aneurysms which generally form on small vessels less than $\frac{1}{80}''$ in diameter, and which themselves vary between $\frac{1}{25}''$ and $\frac{1}{125}''$ in diameter. These have been shown by Charcot and Bouchard to be very common on the cerebral vessels of elderly people; and the rupture of one or more of them is now known to be a frequent cause of the cerebral hæmorrhages which are so common in elderly people. Secondly, there are larger aneurysms which have been long known to occur on one or other of the larger arterial trunks at the base of the brain, or on some of the main branches of the circle of Willis as they lie in the pia mater on the surface of the brain. These may vary in size from a pea to a small walnut. Aneurysms of this size may also very rarely be found within the brain substance.

With miliary aneurysms we are not now concerned. They do not of themselves produce morbid symptoms, and their presence is pathologically or genetically related to the intra-encephalic hæmorrhages we have already considered.

We are now concerned with the larger aneurysms above referred to; but only, however, with these in the period anterior to their rupture, when, like a tumour or other adventitious product within the cranium, they are capable, by their mere presence and by the pressure which they occasion, of giving rise to paralytic or other symptoms. It is only some of such aneurysms that manifest their presence in this way, that is, by the production of any appreciable symptoms anterior to their rupture.

Whenever the rupture of such an aneurysm takes place, the clinical symptoms with which we are confronted are, of course, merely those of cerebral hæmorrhage, which, in the majority of cases, is meningeal, and in a small minority only of intra-cerebral type. Hæmorrhage into the cerebellum from this cause is, up to the present date, unknown.

The largest meningeal aneurysm that the writer has seen or read of is to be found in the museum of University College. This is an aneurysm on the basilar artery, nearly globular in form and about $\frac{7}{8}''$ in diameter. An aneurysm of such a size, in such an important situation, would almost certainly have given rise to some definite symptoms during life; but where we have to do with a smaller aneurysm, about the size of a pea or a small bean, on the middle cerebral artery within the Sylvian fissure, this arterial dilatation may have been slowly formed in such a situation without giving rise to any appreciable symptoms. Yet this is about the commonest situation for such an aneurysm to occur, and it will only rarely be found to have attained a size larger than that above mentioned.

On the other hand, the largest intra-cerebral aneurysm which the writer has ever seen or read of, he found in one of his patients who died from a profound apoplectic attack lasting about four hours. There was an enormous effusion of blood which had burst its way from the right corpus striatum into the lateral ventricles. This was found to have proceeded from a ruptured aneurysm about the size and shape of a small chestnut, which had been lodged in the substance of the lenticular nucleus of the striate body. Even in this case no antecedent hemiplegia seems to have existed; so that aneurysms about the size of a small bean or a small filbert, which are of about the largest size likely to be encountered within the cerebral substance, will probably, in the majority of cases, give rise to no very definite symptoms antecedent to their rupture, unless they occur in some altogether exceptional situation. The writer has, for instance, seen paralysis with extreme atrophy of one half of the tongue occur in an elderly patient as the sole clinical result of a fusiform dilatation with great thickening of the external coat of one of the vertebral arteries. The corresponding ninth nerve as it crossed this vessel had become organically adherent to it, and had here undergone a process of complete sclerosis.

From what has been said above, it seems clear that it is only under extremely rare and exceptional conditions that a meningeal or an intra-cerebral aneurysm manifests its existence by any definite paralytic symptoms anterior to the period of its rupture.

These aneurysms may occur at any period from childhood to old age; their occurrence at the earlier periods of life being, as suggested by Church, often explicable by reason of the previous existence of embolism in the same site. This being so, we should have some ground for expecting that larger aneurisms in children or young adults would occur principally in the middle cerebral or in one of its branches, and more frequently on the left than on the right side—in this respect following the order of frequency which obtains for embolism itself.

One other kind of aneurysm within the skull remains to be mentioned, viz., that which occurs on the carotid artery within the cavernous sinus or just before it divides into the middle and anterior cerebrals. The existence of such a dilatation is rare, but when it occurs the aneurysm is likely to produce some definite symptoms during the later stages of its formation, and these may of course exist for a long time before a fatal termination is brought about. In such a case we may especially expect to meet with nasal or lateral hemiopia together with paralysis of one or more of the ocular nerves, on the side affected.

IX. HYDATID CYSTS.

Two kinds of Cestoid Entozoa are met with in the human brain in their immature or larval condition. They are the hydatid- or echinococcus-cyst, which represents the second or 'scolex' stage in the development of Tænia echinococcus, an entozoon infesting the intestines of the dog; and the cysticercus which is the equivalent developmental stage of T. solium (the pork worm), and T. mediocanellata (the beef worm).

In reference to our subject, hydatids are decidedly the most important of the two parasites. They are far more apt to produce paralysis than cysticerci, while the latter are much more prone to be associated with convulsions.

In the majority of cases only one hydatid cyst is met with. In size it varies from that of a marble to a large orange, though occasionally even this latter size may be notably exceeded. Thus, in a case reported by Abercrombie, an immense hydatid cyst was found within the left lateral ventricle, which nearly extended to the circumference of the brain on the same side, and contained about sixteen ounces of fluid. In another case recorded by Rendtorff a mass of hydatids weighing two and a half pounds was found in the same situation in a girl only eight years of age. The hydatid cyst is not unfrequently lodged in the midst of the white matter of one of the hemispheres, and it may increase in size till it occupies almost the whole of one of the lobes—anterior, middle, or posterior as the case may be. Occasionally it may occupy the greater part of two contiguous lobes and project towards the circumference as well as into the lateral ventricle. I have only found one case recorded in which the hydatid cyst was lodged in the substance of the cerebellum; in this instance the cyst was rather large, it measured 3″ by 2″, stretched across from the right to the left lobe, and projected into the fourth ventricle.

Rarely two, three, or more hydatids are met with in different parts of the same brain. In these cases, their size is generally in inverse proportion to their number; they may vary between that of a mustard seed and a hazel nut. One case of this kind is referred to by Davaine in which many hydatids were found in the meninges and at the surface of the brain, while others existed in the corpus callosum, in the left parietal lobe, in the right optic thalamus, and in other parts.

The duration of the malady naturally varies according to the situation and number of the hydatids; so that their small size where they are multiple may be explained by the more serious nature of the malady and the consequent earlier death of the patient. The increase in size of the hydatid cysts within the brain probably takes place more slowly than it does in other organs, because it so happens that in this organ, the hydatid commonly presents itself as a barren cyst (the so-called acephalocyst of Laennec). As it increases in size no very notable change is generally observable in the surrounding brain tissue, which seems to atrophy as it becomes pressed upon by the growing cyst. Softening rarely occurs in the surrounding brain tissue. After the cyst ceases to grow a capsule of connective tissue may form round it, though in the brain any such investing capsule is usually thin.

In regard to the actual duration of the malady, this probably varies much in different cases, as I have already intimated. Davaine refers to one case in which, from the clinical evidence, it seemed almost certain that a large single hydatid cyst must have been at least four years old.

Hydatids in the brain seem to occur as often in the one sex as in the other. As regards age they appear to be met with in the great majority of cases in individuals between the ages of ten and thirty. Thus out of thirty reported cases (the references to which are given in Reynolds' 'System of Medicine,' Vol. ii., p. 499), the writer has found the age stated in 24; of these 3 were below 10 years of age (5, 7, and 8 years), 3 above thirty years ('middle age,' 37 and 38 years respectively); while the remaining 18 were between the ages of 10 and 30 inclusive. This is very notable, and in striking contrast with what is known concerning the Cysticercus and its tendency to occur rather in the latter half of life than in younger individuals. These facts seem to point to the possibility of the more frequent direct infection of children and young people through the dog's tongue with ova of Tænia echinococcus; though why older people should be more prone to swallow the ova of the two common human tape-worms is more difficult to explain even in a tentative fashion, partly because we are so ignorant as to the most common channels or vehicles by which these latter ova are taken into the alimentary canal. It would be out of place here, however, to discuss this problem.

Hydatid disease is fortunately rare in this country, so rare indeed that in a given case presenting symptoms pointing to the existence of some tumour or other adventitious product within the skull one would scarcely be justified in arriving at the diagnosis of 'hydatids within the cranium' except in the face of more positive evidence that hydatids were present in other parts of the body. In countries like Iceland, or even in certain parts of South Australia, where hydatid disease is very prevalent, such an amount of reserve would not be needed, since hydatids might be nearly, if not quite, as common there within the cranium as any other adventitious product.

X. CYSTICERCI.

In the brain, Cysticerci vary in size from a pea to a small horse-bean or almond. The serous cysts in which, in other situations, they are usually enclosed, are often absent, so that they are bounded only by a smooth layer of slightly compressed brain substance. They generally exist in large numbers in the same brain, and are very rarely solitary. From 10-20 are frequently met with. Cruveilhier recorded a case in which more than 100 were found within the cranium of the same individual, and of these about 50 were lodged in the cerebellum. They may be found in almost all parts of the brain, but, speaking generally, they are by far the most abundant at or near the surface of the brain, or in close connection with the grey matter of the convolutions. They are extremely frequent in the pia mater where they press upon and partially imbed themselves in the surface grey matter. They have likewise been found in the midst of the white substance of the hemispheres, in the central ganglia, in the pons, in the crura cerebri, as well as in the cerebellum. Real cysticerci have also been found in the choroid plexuses of the lateral ventricles; though here they have to be carefully discriminated from the small simple cysts which are so common in this locality.

Cysticerci occur with about equal frequency in the two sexes. They may be met with at all ages beyond infancy, though they occur most frequently during the latter half of life, and occasionally they have been found in very old people.

Owing to the comparative smallness and plurality of cysticerci, combined with their frequent situation in or about the cortex of the brain, it is well to recollect that these particular intra-cranial adventitious products are especially apt to be associated with convulsions, and often with mental impairment of a marked character. Paresis, rather than paralysis, may coexist with these symptoms in some of the cases.

XI. SIMPLE CYSTS.

In some very rare cases simple cysts have been found in the cerebellum and other parts of the brain by Pye-Smith, Hadden, and others. These cysts have not been hydatids—nor have they been secondary pseudo-cysts formed in the site of an old hæmorrhage, or in the substance of some pre-existing sarcoma or glioma. They have been commonly associated with the presence of similar cysts in other organs of the body, such as the liver, the kidneys, the pancreas, &c. The symptoms have been of a general character, indicative of the presence of an intra-cranial growth or abscess. A more special diagnosis would scarcely have been possible in any of the recorded cases.

XII. CONGENITAL OR EARLY INFANTILE PATHOLOGICAL STATES OF THE BRAIN.

Pathological processes may take place in the brain during intra-uterine life which, later on, may be followed by atrophy and arrest of development in the part affected. In other cases during the act of parturition pathological accidents may occur within the cranium (and of these latter by far the most common are arachnoid hæmorrhages), which again may entail a certain amount of atrophy, or more correctly speaking arrest of development on the half of the brain pressed upon.

The result in these two classes of cases is very similar, viz., the production of a more or less complete hemiplegic condition associated with more or less of mental defect and other symptoms, coupled with a diminished rate of growth in the paralysed limbs, so that with increasing age (up to the period of the attainment of full stature), the disproportion in size goes on increasing between the non-affected limbs and those which are paralysed. This smaller size and diminished rate of growth in the paralysed limbs is generally more marked in the arm than in the leg; and is commonly associated with more or less marked rigidity or contracture about the affected elbow, wrist, and fingers.

These constitute a very typical class of cases when associated with a history that the condition dates from birth. It must be remembered, however, that a practically similar condition may be induced when severe cerebral lesions in the form of hæmorrhages or softenings occur in infancy or early childhood—that is, up to the fifth or sixth year or sometimes even a little later. Where hemiplegia occurs at this period of life as a result of some severe lesion, this may entail an atrophic process in the corresponding region of the brain, and the limbs on the opposite side of the body are, through this brain injury, deprived (in one or other way) of a certain amount of their proper nutritive energy, and, as a consequence, they grow at a much slower rate than those of the other side of the body.

Later in childhood than the period named above, it is more rare for cerebral lesions of equal size to have such a profound effect upon the nutrition of the paralysed limbs; and even if a correspondingly depressant effect were produced upon the rate of growth in the affected limbs, the fact that at the date of the later lesion the growth of the body was more advanced, would of itself leave less opportunity for differences in the rate of growth on the two sides of the body to show themselves.

These cases are referred to here merely for the sake of clinical convenience in order to call attention to the effects produced by severe lesions of one cerebral hemisphere in very early life.

The lesions do not constitute a group apart from those already considered. They are mostly hæmorrhages or softenings, and their sequelæ; and among such sequelæ there are two which stand out in especial prominence—viz., atrophic processes and processes of sclerosis.

The atrophic processes occur partly as immediate and proximate results of the primary lesion, and partly as more remote results gradually developing themselves. Among the most interesting of these secondary atrophies is that which occurs in the lateral lobe of the cerebellum opposite to the cerebral hemisphere affected.

Of the sequential processes of sclerosis, some limit themselves pretty closely to the confines of the primary lesion, while others, taking their start there, may subsequently go on so as to implicate a considerable surrounding area of the brain substance or even the whole remaining part of the hemisphere affected. In some very rare cases the nature of the initial lesion is extremely difficult to detect, and yet one hemisphere may be sclerosed throughout and not more than one-third of the bulk of its fellow of the opposite side. Such a case I have recently had the opportunity of examining, in which the paralysing lesion (whatever it was) occurred abruptly and was associated with a hemiplegic form of paralysis at the age of four years, the child having previously been quite healthy. In this case, which was a typical example of the group to which I am now referring, the patient, who was an imbecile, lived to attain the age of sixty years.

The only cases likely to be confounded with those to which we have just been referring, are the comparatively rare cases of acute spinal paralysis in young children in which one arm and one leg only have been implicated, and that on the same side of the body. The exercise of a little care, however, will easily enable the practitioner to discriminate between these two conditions, which are etiologically so very different. In the latter form of disease, the muscles either do not respond, or respond with difficulty, to Faradism, the leg is generally more paralysed than the arm, and there is no trace of a facial paralysis on the same side; whereas in the cerebral affection, the muscles respond readily to Faradism, the arm is commonly more paralysed than the leg, and there may be still some slight amount of facial deviation.

PATHOLOGICAL DIAGNOSIS.

CLINICAL INDICATIONS FAVOURING THE EXISTENCE OF THIS OR THAT CAUSATIVE CONDITION.

The initial periods of paralyses of encephalic origin are often more or less shrouded by states of insensibility or Coma, some of which are known by the name of Apoplexy—or more definitely as Cerebral Apoplexy. Seeing, however, that this condition of Coma is one which may be produced in many different ways; and that in accordance with such differences in mode of production some of them are, and some are not, apt to be associated with paralysis of limbs, it is evident that a brief preliminary consideration of the causes, and modes of distinguishing, the different kinds of Coma (so far as this may be possible) becomes necessary.

The problems of Pathological Diagnosis have therefore to be studied under two main heads:—(A) as they present themselves during the Apoplectic Stage, and (B) as they present themselves after or in the absence of such a stage.

When a medical man is first summoned to a patient the latter may be in a comatose condition, and his first object then must be to decide as to the cause of the patient's insensible condition—a problem which in many cases is found to be one of extreme difficulty, especially where the patient has been found in that state, and where nothing is known as to his antecedents, or at least as to the events more or less immediately preceding his being found in an insensible condition. On the other hand, a doctor even more frequently sees a paralysed patient for the first time at a period more or less remote from the onset of his illness, when he can often only obtain more or less vague details as to the mode in which it has commenced. The consideration of the problems pertaining to a pathological diagnosis in cases of paralysis of encephalic origin is, therefore, naturally divisible in the manner above indicated.

(A). PATHOLOGICAL DIAGNOSIS IN THE APOPLECTIC STAGE.

Comas or comatose conditions are due to very various causes, but for our purpose they are primarily divisible into two groups. Such a condition may, in certain cases, (1) show itself as it were abruptly and without antecedent illness of a definite kind; or, on the other hand, (2) it shows itself in the course of some malady known to pre-exist and known to be liable to such an association.

PRIMARY COMA.

1. External violence causing shock or laceration of brain.
2. Alcoholic Intoxication.
3. Narcotic Poisoning.
4. Sunstroke or prolonged exposure to Cold.
5. Encephalic hæmorrhage.
6. Cerebral Embolism.
7. Cerebral Thrombosis.

SECONDARY COMA.

8. Post-epileptic coma.
9. Coma in General Paralysis of the Insane.
10. Coma with Meningitis.
11. Coma with Tumour or Abscess of the Brain.
12. Uræmic coma.
13. Diabetic coma.
14. Coma with Acute Yellow Atrophy of Liver.
15. Coma with Hyperpyrexia.

1. External Violence causing Concussion or Laceration of Brain.—Under this head are included the most diverse injuries met with in times of warfare, produced by various weapons or missiles; and also the effects of accidental or purposive blows, or falls, such as happen only too frequently to persons of all ages.

In these various cases the fact of the external injury is generally obvious; but when a person is found in an insensible condition, presumably of traumatic origin, the point that must often for a time remain doubtful is, whether (1) the person is merely suffering from the effects of shock or concussion; or, whether (2) there is in addition more or less of actual damage to the cranium or its contents.

In this latter case we may have to do with (a) fracture of the skull, (b) meningeal hæmorrhage, (c) hæmorrhage into the substance of the brain, (d) lacerations of the brain substance, or (e) a combination of any two or three of these conditions.

In some cases time must be allowed for the effects of mere shock to subside before it is possible to form a positive judgment as to the existence or not of laceration of the brain, of hæmorrhage into its substance, or of a hæmorrhage pressing upon its surface. It is well to bear in mind that in some cases, apparently trivial accidents may lead to the gravest consequences. Thus, a man may merely slip in the street, and fall, striking his head against the pavement, and yet with so much force as to cause not only lacerations of the brain substance but extensive meningeal hæmorrhages, followed by speedy death (Path. Trans., Vol. xvii. p. 10).

In other cases, however, it may be obvious from the first that some such serious injuries exist over and above the effects of mere brain shock. This is the case, for instance, when signs of facial paralysis exist on one side, and when the limbs (one or both) of this side fall, when raised, in a much more flaccid and helpless manner than those of the opposite side; also when unilateral rigidities or clonic spasms occur on one side, or unequally on the two sides of the body, or when conjugated deviation of the head and eyes exist, or when one pupil is contracted and the other is dilated. These are signs recognisable during the stage of coma, and the presence of two or more of them will certainly indicate the existence of some amount of organic mischief within the cranium.

Again, another question must present itself in some of the cases in which we are called to a person who has fallen and is picked up in an insensible condition; and that is, whether we have to do with the mere effects of the fall (whatever their degree of severity), or whether the person was taken ill first, with some sort of fit, and fell only in consequence of such fit. If the person fell in consequence of an epileptic attack, the only complication might be that such a person is apt to remain in a condition of stupor or insensibility for a longer period than he would have done as a mere consequence of the fall. On the other hand, if it were the onset of an ordinary apoplectic attack which caused the person to fall, the case is necessarily a much more serious one; for not only may the lesions causing the apoplexy be more or less aggravated by the fall, but we should, in that case, have to do with a patient suffering from the effects of such lesion over and above the effects of independent injuries that may have accrued from the fall itself.

2. **Alcoholic Intoxication.**—A man or woman is picked up in an insensible condition in the street, with the breath smelling more or less strongly of spirits, and the facile conclusion of the policeman is that

he has merely to do with a case of drunkenness. Again, a person may have been seen to be reeling about, and apparently intoxicated in the street, before he or she has fallen or been knocked down by some passing vehicle; in the latter case it is possible that the person may be brought by the police to the casualty room of some hospital. The house surgeon should bear in mind that in such a case he is not called upon, nor should it be expected of him, to decide at once, after his first examination, that he has to do merely with a case of drunkenness. Drunkenness may be present, but there may be something much more serious besides, the existence of which is apt at the time to be more or less completely masked by the alcoholic intoxication. In all such cases, time ought to be allowed for any mere drunkenness to subside, and with this object in view the person ought to be allowed to remain in a bed attached to the casualty room, for the necessary number of hours, before the surgeon positively decides as to the absence of brain injury or disease and permits the person to be handed over to the police.

The adoption of such a rule greatly diminishes the chance of a mistake, which may be very harmful to the person principally concerned; and also avoids the public scandal which is caused by the announcement in the papers that a person who had been taken to this or that public hospital, had been sent away from that institution, had died the same night or the next day in a police-cell, and that a post-mortem examination had revealed the existence of serious disease or injury of the brain. Newspaper paragraphs headed 'Drunk or Dying,' would, in short, become decidedly less frequent.

Not only may the effects of disease sometimes simulate various degrees of drunkenness very closely, but, on the other hand, obvious drunkenness may for a time, as above indicated, more or less completely mask the signs of some important injury or disease of the brain. These are facts which should always be borne in mind by the medical man.

An excessive quantity of alcohol induces stupor or coma, often with flushed face and dilated pupils. If vomiting does not occur or the stomach-pump be not used, symptoms of collapse may, in rare cases, speedily set in, where the amount taken has been extremely large.

After the preceding remarks, it may be sufficient to indicate that in cases brought before us connected with excess of alcohol, we may have to do with one or other of the following conditions:—

(1). Alcoholic intoxication, alone.

(2). Alcoholism, plus a fall or blow, but no intra-cranial damage.

(3). Alcoholism, plus a fall or blow, together with more or less serious intra-cranial damage.

(4). Alcoholism (more or less), plus a more or less severe apoplectic attack due to intra-cranial hæmorrhage or vascular occlusion.

These several conditions must be discriminated from one another by the most careful examination of the person at the time when he is first seen, and, in all cases where there is room for the least doubt, no definite opinion should be at once expressed, but the person should be kept some hours under observation, and examined again at a later stage when the clouding effects of alcoholic excess have had time to wear away.

3. **Narcotic Poisoning.**—Only a few remarks need be made here on the subject of narcotic poisoning. The consideration of alcoholic intoxication should really have come under this head, as alcohol is one of the narcotic poisons. It was thought best, however, for practical purposes, to deal with this subject separately.

Of the remaining narcotic poisons there are but three whose poisonous effects are at all commonly met with, viz., opium and its derivatives, chloroform, and prussic acid or its compounds. Of these poisoning by opium or its derivative morphia, is by far the most common; poisoning by the other two drugs are comparatively rare events.

The circumstances under which narcotic poisoning occurs are divisible into two distinct categories. Thus we may have,

(a) Cases in which suicide has been attempted.

(b) Cases in which a medicinal overdose has been carelessly administered, or unwittingly taken.

(a). The cases coming under this category often present no difficulty in the way of diagnosis, since the person who commits suicide commonly takes no precautions to conceal the means by which the attempt upon his or her life has been made. Near at hand, therefore, a bottle may be found which by its label, its remaining contents, or by its odour may testify as to the meaning and cause of the person's condition.

Prussic acid was formerly most apt, or rather a bottle which had contained it, to be found under such conditions. Now that such poisons as this are less easily obtained by the public, poisonings by prussic acid are becoming decidedly less frequent. Still suicide is occasionally effected or attempted by one of its compounds, potassium cyanide, a highly poisonous salt, commonly employed by photographers and workers in electrotype, in the exercise of their calling. In either of such cases the breath may have the distinctive odour of prussic acid. The person is insensible; the skin is commonly cold and dusky; the pupils are dilated; the breathing is shallow and irregular in character; the pulse almost imperceptible; spasmodic closure of the jaws and perhaps actual convulsions may also occur. Where large doses are taken death may occur in fifteen, ten, or even five minutes.

Opium and morphia are not so commonly taken by persons with suicidal intentions; and chloroform is almost never had recourse to with such an object.

(b). Inadvertent deaths from chloroform inhaled by the persons themselves, on their own responsibility, for the relief of some pain, have unfortunately been only too frequent in recent years. Some lamentable cases have even occurred in which chloroform has been rashly self-administered by medical men with the view of courting sleep, with the result that they have been overtaken by the sleep of death. Sometimes again, a poisonous dose of chloroform has been swallowed with the view of procuring sleep.

In all these cases death may occur before the patient is found, but should this not be the case, the chloroform bottle is commonly near to tell its tale, or the patient's breath may smell strongly of chloroform, whilst he is found to be in a profoundly comatose condition; with widely dilated and insensible pupils, pulse slow and full (with body warm), or slow and feeble (with surface cold); the respirations being hurried and slightly noisy, or weak and slow.

Poisoning from prussic acid itself or from potassium cyanide does not often occur from inadvertence. It does happen, however, rarely from the eating of such vegetable products as bitter almonds, or the kernels of peaches, apricots, or other stone fruit. All these contain variable amounts of prussic acid, and when they have been consumed too freely have occasioned serious signs of poisoning. The leaves of the laurel used in flavouring also contain much prussic acid, and when they have been too freely employed poisoning has sometimes occurred. The symptoms in all these cases are of the kind described in the previous section (a).

The cases of inadvertent poisoning by opium or morphia are unfortunately only too common, and particularly as a consequence of the careless administration of various patent medicines, so-called "soothing syrups," containing opium or morphia—the victims here with especial frequency being infants or young children, who are peculiarly sensitive to its influence. Adults, again, who are affected with renal disease may sometimes exhibit marked symptoms of opium poisoning after doses of opium or morphia, which would produce no poisonous symptoms in persons in an ordinary state of health. The increasing susceptibility with old age to the influence of opium, as well as the occasional existence of an idiosyncrasy rendering the individual at all periods of his life extremely vulnerable to the influence of opium or morphia, are other additional causes why poisoning by such pain-killing drugs as these is apt to occur not unfrequently from carelessness or mere inadvertence.

However it may have been brought about, and at whatsoever age the poisoning by opium or morphia may occur, the patient presents usually a well-defined and typical condition.

Drowsiness and stupor soon lapse into insensibility or coma, from which it may be difficult to rouse the patient even for a moment. The pupils are strongly contracted (often to pin-point size) and quite insensitive. The breathing gradually becomes first slow and then stertorous, the pulse weak, and the face inclines to lividity. The surface generally may also be cold and livid, or bathed in sweat. All the limbs are relaxed. Vomiting sometimes occurs; and occasionally death is more or less immediately preceded by convulsions. In very rare cases the pupils are said to have been dilated.

It is most important to bear in mind the fact that hæmorrhage into the pons Varolii produces a form of apoplexy which is liable to be, and has in fact been at times confounded with opium poisoning. In each case there may be a comatose condition, with pupils contracted almost to a pin-point size, with slow stertorous breathing and disordered pulse, together with general muscular relaxation. It has been the condition of the pupils especially which has misled or thrown off their guard those who have fallen into this serious error of diagnosis. Those who are aware of the resemblance in question may generally detect in a case of hæmorrhage into the pons Varolii the coexistence of other signs, such as the existence of some unsymmetrical condition about the face, or some slight rigidity or twitchings perhaps in the limbs on one side, which are not, on the other hand, likely to be met with in a case of opium poisoning.

4. Sunstroke or Prolonged Exposure to Cold. These are causes of a comatose condition which should not be forgotten. Sunstroke would be better known as 'heat-stroke,' since undue exposure to heat from any cause, especially in debilitated or exhausted states of the body, is liable to give rise to the conditions commonly known as 'sunstroke,' independently it may be of direct exposure to the sun.

There are two forms of this affection which may give rise to a condition of insensibility simulating that from cerebral hæmorrhage, viz., the 'asphyxial form' and the 'hyperpyrexial form' of Fayrer. Speaking of the former variety he says,—"The symptoms of this form of insolation, the real *coup de soleil*, are those of sudden and violent injury to the nerve centres—unconsciousness and cold skin, feeble pulse, all the symptoms of depression; death resulting from rapid failure of the respiration and circulation." Occasionally, during this state, actual cerebral hæmorrhage of more or less magnitude may occur, and thus complicate the clinical picture.

The hyperpyrexial form of insolation gives rise to a condition in all respects similar to the hyperpyrexia that is occasionally apt to occur in the course of rheumatic fever and other acute febrile diseases, where a comatose condition is produced in which the temperature rapidly rises to 108° or even 110°.

The rapid rise of temperature, associated with a hurried gasping respiration, a pulse full and slow or quick and jerking, great restlessness, with pungent heat of skin and frequent micturition, is the condition commonly met with in these cases. The head and neck are extremely congested—more or less livid—and the pulsations of the carotids are visible. The condition of the pupils is not distinctive; they may be contracted at first and subsequently widely dilated. Coma soon becomes marked; and convulsions may occur before the patient dies, asphyxiated. With a temperature reaching 108°, recovery becomes an extremely rare event.

Exposure to intense or prolonged **cold** is in some countries an occasional cause of a comatose condition. The circumstances in which the patient is found will, in such cases, often of themselves suffice to make the diagnosis clear. Should relief come in time such a comatose condition may be completely dissipated under suitable treatment.

The symptoms produced by extreme cold are said to be these—"the propensity to sleep becomes irresistible; everything grows strange; the senses are confused; the mind grows dull, the ideas incoherent, and the speech stammering or raving; respiration, at first interrupted, becomes slow; the heart's action is feeble, quick, hard, irregular, and sometimes painful, and the pulse progressively smaller; the pupils dilate; the brain becomes stupefied; and finally deep coma indicates the approach of inevitable death." (Durham, Quain's 'Dictionary,' p. 271). The early symptoms might be mistaken for those of alcoholic intoxication.

5. Encephalic Hæmorrhage.

6. Cerebral Embolism.

7. Cerebral Thrombosis.

These three pathological conditions are the common causes of insensibility or coma (so-called 'apoplectic attacks') from cerebral disease of idiopathic or non-traumatic origin. They ought always to be diagnosed from one another, so far as this may be possible. At times such a diagnosis cannot be made with any certainty.

The conclusion that in a given case of coma we have to do with one or other of these conditions, will usually have to be made upon general principles and after excluding the existence of other possible causes of the apoplectic state. In this condition it may be very difficult, if not impossible, to recognize any special powerlessness on one side of the body; and in the general muscular relaxation characteristic of the state no lack of symmetry may be detectable about the face. Still these signs should always be looked for, and in some cases we may recognize a greater flaccidity of the cheek on one side, or that the limbs on one side are completely flaccid, whilst those of the other perhaps present some amount of rigidity, or may be the seat of distinct clonic spasms. Inequality in the size of the pupils is another sign which should be looked for, also inequality in the sensitiveness of the two conjunctivæ, so that touching the conjunctiva on one side leads to reflex contraction of the lid, whilst on the other side the same action is followed by no such result. Again we may sometimes recognize that one upper extremity is distinctly warmer than the other; a difference of temperature being occasionally distinctly noticeable in the palms of the hands even within one hour from the commencement of a not very profound apoplectic attack. In such a case, the side on which the temperature is higher is the side on which evidence of paralysis may be looked for, or on which it will become manifest subsequently, should the patient recover from the apoplectic state. But over and above all these in importance there is a sign of the existence of a definite cerebral lesion, of the greatest diagnostic significance when it occurs, and one, moreover, which obtrudes itself upon the attention of any one who is alive to the nature and value of such a sign—I allude to what is known as 'conjugate deviation of the head and eyes.' When it occurs in a non-traumatic apoplectic patient, it may be said to be absolutely conclusive evidence that the condition is due to cerebral hæmorrhage, or occlusion of some of the cerebral vessels by embolism or thrombosis. It is desirable, therefore, to say something in this place concerning such an important sign of the most common form—from an etiological point of view—of the apoplectic condition. It is a sign very far from occurring in all apoplexies of this type; but it occurs in many of them, and when it presents itself it has all the significance above stated.

When *Conjugate Deviation of the Eyes and Head* is encountered in an apoplectic patient to a well-marked extent, the head is seen to be strongly turned to one or other shoulder, whilst both eyes are directed outwards and rather upwards in the same direction—it is as though the patient were making a persistent effort to look over one or other shoulder. Where the condition exists to a less marked extent, there is only a slight deviation of the head to one or other side, though both eyes are strongly deviated in the same direction.

The deviation of the eyes is quite distinct from strabismus, the optic angle being neither increased nor diminished. When there is rotation of the head any attempt to bring it back to its ordinary condition is impeded by the rigid condition of certain muscles. When the effort of the observer to turn it or to maintain it in its natural position is relaxed, the head immediately reverts to its previous position. The muscles of the neck in a state of contraction are mainly those on the side towards which the head is turned, though the opposite sterno-mastoid is also in an associated state of contraction. In the slighter forms of this condition the eyes may not be immovably fixed in the one direction; the patient may (especially when semi-conscious) be seen to move them in attempts to look at a bright object, perhaps as far as the middle line, but not beyond. He generally cannot look in the least towards the opposite side, and when the stimulus is withdrawn the eyes at once return to their original position.

Although this condition had been previously noted and referred to by several writers, it was first completely studied by Prevost, who published an important memoir on the subject in 1868.

Conjugate deviation of the head and eyes is a condition which very rarely shows itself with a hemiplegia of gradual onset; it occurs especially in association with cerebral hæmorrhage or with either mode of occlusion of cerebral vessels. It is, therefore, one of the initial symptoms in a hemiplegia setting in with an apoplectic or sub-apoplectic stage, and when death occurs in twenty-four to forty-eight hours it may persist to the last. In cases, however, in which the apoplectic condition clears up, and the patient is left simply hemiplegic, it commonly disappears gradually in the course of a few days; though sometimes it persists much longer—for weeks or months.

The investigations of Prevost led him to believe that the appearance of this sign was not necessarily connected with disease in any particular part of the cerebrum, but he came to the important conclusion that in the class of cases to which we have just been referring there was an all-important constancy in the direction of the deviation. In all cases where the lesion is situated in one cerebral hemisphere, the patient, he says, looks towards that hemisphere which is the seat of lesion.

It is this definiteness of indication that gives the sign its value, in the first place, for the diagnosis of apoplexy itself, and secondly for the information which it is capable of affording as to the side of the brain that is damaged, at a time when no other sign may be forthcoming even as to the existence of a hemiplegic condition. I have known this sign also give most valuable indications in such a case as this—a patient who is already hemiplegic, but quite conscious and going on well, more or less suddenly lapses into an apoplectic condition, but shows a conjugate deviation of the head and eyes in the direction of the previously paralysed limbs. Now to a person not knowing the exact significance of the latter sign, the most natural supposition would be that the apoplectic condition was due to some extension of the disease in or near the old focus. But to a person knowing its real significance, the indication would clearly be that something much more grave had occurred—that is, that the previously sound cerebral hemisphere had now become the seat of disease. And, with lesions in both hemispheres, the patient's condition would be rendered much more helpless, both mentally and physically, even should he survive the additional perils attaching to such an apoplectic state.

Prevost admitted only one exception to the rule above stated, and that was in regard to lesions occurring in the pons Varolii, when the deviation was sometimes found to be towards the side of lesion and sometimes towards the side of paralysis. Desnos subsequently maintained that in lesions of the pons the deviation was always towards instead of away from the paralysed side. But, as we shall now see, the views of Prevost as well as of Desnos stand in need of some qualification.

This subject has more recently been studied principally by Landouzy and by Grasset, and they have shown the extreme importance, in reference to this sign, of distinguishing between the effects of paralysing and of merely irritative lesions. The clue to this important extension of our knowledge was furnished by the experimental observations of Hitzig and of Ferrier.

It is now a matter of common observation that lateral deviation of the head and eyes occurs very frequently with the onset of some kinds of epileptoid attacks; and where unilateral convulsions are thus associated with conjugate deviation of the head and eyes, it has always been found that the deviation takes place towards the side of the body which is convulsed, and therefore away from the side of lesion. If we were to enter into the full physiological explanation of this sign (which would be out of place here) it would not be difficult to show that this result is just what might have been expected. But without entering into any elaborate illustration it may easily be shown from the analogy of what takes place when the muscles of one side of the face are paralysed and convulsed respectively, that an irritative lesion producing lateral deviation of the head and eyes ought to give rise to quite a different result from that which would follow upon the establishment of a paralysing lesion in the same part of the same cerebral hemisphere.

Where a left hemiplegia is caused by a destructive lesion in the right hemisphere we find the lower part of the left side of the face paralysed and the right angle of the mouth consequently pulled slightly outwards and upwards (that is towards the side of the brain lesion), owing to the unbalanced action of the muscles on that side. Just as we have the right angle of the mouth pulled upwards and outwards in a case of left hemiplegia, so, when a conjugated deviation of the head and eyes is associated with a left-sided paralysis, the deviation takes place towards the right side, by virtue of certain unbalanced muscular actions similarly tending to draw the head and eyes in this direction.

Where, on the other hand, we have to do with left-sided unilateral convulsions caused by an irritative lesion in the right hemisphere, we now find that muscular action is in excess on the left side of the face, and as a consequence the left angle of the mouth is drawn upwards and outwards—that is to say the mouth is drawn towards the side which is convulsed. But supposing the irritative lesion to occur in certain regions of the brain, presently to be referred to, then, in association with left unilateral convulsions we may have a left-sided deviation of the head and eyes—that is the head and eyes are now turned away from the side of brain lesion and towards the side of the body which is convulsed. The deviation of the head and eyes follows the same rule as that which applies to the face; it is, therefore, only natural that we should have in the case under consideration a deviation in a direction the reverse of that which obtains in the case of a left-sided paralysis. If in the latter case the deviation is towards the side of lesion because of the loss of power in a certain set of muscles; then it is only reasonable to expect that when these same sets of muscles are, by an irritative lesion, called into excessive action, a deviation should occur in the reverse direction.

Thus, in cases of lesions in the cerebral hemispheres, the old rule laid down by Prevost requires to be modified, as it has been by Grasset, in accordance with the following terms:—When conjugate deviation exists with a lesion in one of the cerebral hemispheres, the patient looks to the side of convulsion when this exists, and looks in the direction of his lesion when there is paralysis and no convulsion.

In the consideration of the effects of lesions of the pons Varolii the same kind of distinction requires to be made between irritating and destructive lesions. The law of Denos in regard to this part of the brain has been found to be too absolute. Prevost was right in saying that with lesions in this region the deviation is sometimes away from and sometimes towards the seat of lesion, only he did not define the conditions under which such variations take place. This has been done, however, by Grasset, who has formulated the following rule:—In lesions of the pons Varolii, where conjugate deviation exists, the patient looks towards his paralysed limbs when there is paralysis, and towards his lesion when there is spasm or convulsion.

Here, again, it would be possible, if this were the place for it, to work out a physiological interpretation that would suffice to explain why this different rule should obtain in regard to lesions in the pons Varolii, from that which holds good for lesions in the cerebral hemispheres themselves. But, as it is, we can only hint at the kind of explanation that would be given, and refer those who wish for further information to the ingenious exposition of this side of the subject which is given by Dr. Ross (" Diseases of the Nervous System," Vol. i.). One of the principal physiologico-anatomical conditions upon which conjugate deviation of the eyes and head depends, is the fact that in the two eyes there is an associated activity between the external rectus of the one and the internal rectus of the other, and consequently between the nucleus of the sixth nerve and a part of the nucleus of the third nerve. There is reason to believe that this associated activity of part of the nucleus of the third nerve, is aroused through commissural fibres coming to it from the nucleus of the sixth, so that when a person looks towards the left side, the excitation of the left external rectus muscle carries with it as a necessary consequence the excitation of the right internal rectus (as it also entails, in a more indirect way, the activity of the group of muscles needful to rotate the head to the left side). Thus in looking to the left or to the right side, the initial and principal activity to be considered is that of the nucleus of the left or of the right external rectus respectively. But a lesion in the pons Varolii, especially in its lower half (Fig. 15), may affect the roots of the sixth nerve, just as they may affect the roots of the facial nerves, directly (that is, anterior to the decussation of the fibres of each on their way to the opposite cerebral hemisphere). Consequently, just as, in lesions of this part of the pons Varolii, we meet with a paralysis of the face on the side opposite to that on which it occurs when we have to do with a lesion of the cerebral hemisphere on the corresponding side, so, may we expect that, with a lesion in the lower part of the pons on one side, the direction of deviation of the eyes and head (when it occurs) would be the reverse of that which obtains for a lesion higher up in the cerebral hemisphere on the same side. And, if the direction of deviation is reversed for paralysis, so would it be reversed in cases in which there is an irritative rather than a destructive lesion operating on one or other side of the pons Varolii.

What can be said concerning the localising indications afforded by this sign, of the existence of disease in regions other than the pons Varolii, and especially in cases where disease affects the cerebral cortex, will be subsequently referred to in a separate section (p. 96).

In previous sections we have already discussed the conditions of age, sex, associated pathological conditions, and states of the general health that may be considered to favour the diagnosis of one rather than the other of the three pathological causes of the apoplectic condition at present under consideration. We have now, therefore, only to look to the state of insensibility itself (its clinical characters, that is), together with its mode of commencement, in search of additional characteristics which may guide our judgment as to the exact nature of any individual case that presents itself—whether, in short, it is caused by cerebral hæmorrhage, by embolism, or by thrombosis.

In the first instance we must, of course, eliminate the causes of insensibility or coma already discussed—we must make sure that we have not to do, in the case before us, with the effects of external injury, alcohol, narcotic poisoning, sunstroke, or exposure to cold. If the individual has been seized in the midst of a state of fair previous health, the problem is narrowed to the one with which we are now immediately concerned, that is, whether we have to do with a case of cerebral hæmorrhage, with one of embolism, or of thrombosis.

Should the attack occur as a secondary or intercurrent affection, we must then be able to eliminate all the causes of coma enumerated under our second heading (p. 73) before we could be free to narrow the inquiry to the determination of the existence of one or other of the three common pathological accidents named above.

Where the insensibility has been of abrupt onset in a person over forty and is very profound, or has already existed some hours, the chances are strongly in favour of the apoplectic attack being due to intra-cranial hæmorrhage. Still at times, in intra-cranial hæmorrhage there are premonitory symptoms. Again, the onset of insensibility in such cases may not be abrupt, may not be profound, may not even exist at all.

Where the insensibility is slight, has been of abrupt onset, and occurs in a person under thirty, who has, in addition, a cardiac bruit heard over the mitral or the aortic valves, the chances are largely in favour of the attack being due to cerebral embolism. Still, hæmorrhage does occur not unfrequently in young persons, even in children; and we may occasionally have the coincidence of such a hæmorrhage occurring in a young person who is also the subject of valvular heart disease. It is most important to observe the limitation as regards age. Embolic lesions of the brain occur in the main below forty (especially if we exclude those occurring as a result of ulcerative endocarditis), although organic disease of the valves is only too common in persons beyond this age. The comparative exemption in the later periods of life from embolic occlusion of cerebral vessels is due to the different nature and origin of the valvular lesions occurring in the latter half of life. They are then in a large proportion of the cases of a degenerative type, and not of such a kind as readily to yield embolic fragments; these are supplied principally by the crumbling of such 'vegetations' as are apt to be formed in acute endocarditis, a disease which in its simple form occurs principally in childhood and in youth.

Where the insensibility is slight, and the illness has been of more gradual onset—extending over some hours, or it may be even one or two days,—and where no valvular disease exists, the insensibility and paralysis may be due to thrombosis in one of the larger cerebral arteries. Lately I have seen a well-marked case of thrombosis of the right middle cerebral producing complete left hemiplegia in which there was no loss of consciousness, but mental dulness and confusion associated with pains over the affected side of the head. Similar symptoms might well occur in embolism of the middle cerebral artery—the mental dulness and confusion being produced in each case by the cutting off of the blood supply from a large tract of grey matter with secondary hyperæmia of surrounding parts, whilst the pains over the side of the head may be due to the great disturbance of the blood supply on the affected side of the brain and of its membranes.

It is important to recollect that thrombosis is a process which may occur at any period of life. In young children or in later early life thrombosis in cerebral as in other vessels may occur as an appanage of acute febrile diseases, or during convalescence from such diseases. In women, it may occur within a few days of childbirth, where slight febrile conditions are present. Under the conditions above enumerated, thrombosis occurs as a result of alterations in quality of blood combined with a weakened or irregular action of the heart. During the latter half of life, these cardiac influences co-operate with degenerative and chronic inflammatory changes in the walls of the arteries in bringing about thrombosis. It is as much to be looked for, therefore, in later life as hæmorrhage itself. Chronic inflammatory and degenerative changes in the vessel wall, plus an excited action of the heart favour cerebral hæmorrhage; whereas similar conditions of the vessels associated with a weak and irregularly acting heart, equally favour the process of thrombosis.

The diagnosis between thrombosis and a not very severe hæmorrhage of the ingravescent type is sometimes extremely difficult. Thrombosis of the basilar artery, again, may produce a most profound coma and lead to rapid death. This is the only form of intra-cranial thrombosis in which the symptoms are closely in accord with the more severe forms of apoplexy due to intra-cranial hæmorrhage.

The only other indications to which we can turn for the discrimination of intra-cranial hæmorrhage, cerebral embolism and cerebral thrombosis respectively, during the apoplectic stage are derivable from the study of the temperature of the patient, preferably taken in the rectum. For this means of discrimination we are principally indebted to Bourneville. The pulse and respiration are so variable in different cases as to afford no trustworthy indications for diagnostic purposes, although they are oftentimes of the utmost value for prognosis.

It should here be said that the temperature variations now about to be referred to do not suffice to enable us to decide between embolism and thrombosis. These two modes of vascular occlusion lead to the same kinds of temperature variation, so far as we know at present—just as they lead on to the same kind of pathological sequence, viz., softening of brain tissue. Thus the study of the temperature variations during the apoplectic state will help us to decide between cerebral hæmorrhage and commencing cerebral softening from vascular occlusions. And this will be found to be the most important kind of aid, since it is commonly a comparatively easy problem to decide between embolism and thrombosis upon other and more general grounds, in those cases in which commencing softening is indicated.

Temperature Variations indicative of Intra-Cranial Hæmorrhage. From the point of view of the temperature variations to be met with, Bourneville divides cases of apoplexy due to Cerebral Hæmorrhage into four categories.

(1). Cases produced by very large cerebral hæmorrhages, or in which two or more bleedings rapidly succeed one another. Here death often occurs in an hour or two, and during this period the temperature of the body is decidedly lowered.

(2). Cases in which death takes place in ten to twenty-four hours; where the temperature is at first lowered, though this temporary depression is soon followed by a rapidly increasing rise of body-heat.

(3). Cases terminating in death at the end of a few days. Here there is (a) an initial lowering of temperature; (b) a stationary period in which, after regaining its physiological standard, the temperature of the body undergoes slight oscillations above and below this point; and (c) an ascending period in which the temperature steadily rises.

(4). Cases in which the patient recovers. Here we have an initial lowering of temperature as in the other categories, followed by a temporary rise, and succeeded by a pretty speedy return to the normal standard.

Taking now the cases belonging to the third category, it will be well to state the principal facts ascertained by Bourneville concerning the several thermometric stages that are prone to occur, together with the modifications of pulse and respiration which they entail, or which, at all events, accompany them. The temperatures cited were taken in the rectum, and the variations were studied during each of the three periods—that is, during the period of initial lowering, the stationary period, and the ascending period of body-heat.

(a). *Period of Initial Lowering.*—When the temperature is taken about a quarter of an hour after the onset of the apoplectic attack, it is usually found to be already below the normal standard (probably at about 97¼°), though there may be no notable alteration of the pulse or respiration. Occasionally the fall of temperature takes place a little later, but in either case it may sink as low as 96°. Any recurrence or continuance of bleeding into or upon the brain, causes the temperature to remain for a longer period at this low point, or will again depress it should it have begun to rise. During this period, all the more familiar apoplectic symptoms are well marked—coma, resolution of limbs, stertor with or without occasional vomiting, together with relaxation of the sphincters, are all apt to be met with.

(b). *Stationary Period.*—In an hour or two, should the patient survive, we may begin to get more distinct evidences of the hemiplegic condition. The state of coma passes, perhaps, into one of stupor, in which the patient is less profoundly insensible. The alterations in temperature, however, now to be described may still supervene, even where the coma continues without diminution. During this stage the temperature returns to the normal level for the rectum (99·4°), or it may rise to 101°; and it continues to oscillate rather irregularly between these two points for two, three, or four days. During this time the respirations are not sensibly altered in frequency, and the fluctuations in the pulse are slight and irregular—the rising and falling of the number of pulse-beats being by no means always harmonious with the variations of temperature.

As already stated, in the most severe cases of apoplexy the patient dies in less than two hours—while the temperature is still low. And where the patient is destined to die in from ten to twenty-four hours the comparatively stationary period of body-heat just described is also absent—the primary depression of temperature being followed by a steady and continuous rise, till it reaches at the time of death some point ranging between 104° and 108°. On the other hand, in instances where the stationary period of body-heat is present, if the patient is to recover from the immediate effects of the already existing brain lesion, and if no visceral disease of an inflammatory nature (in the lungs, for instance) should complicate the case, the temperature after two or three days of slight elevation sinks to the normal grade, and there remains. Where, however, the brain lesion is such that the patient is likely to die in the course of a day or two more, the 'stationary period' is followed by another change in the temperature of the body.

(c). *Ascending Period.*—A rise of temperature of a rapid and continuous character, is just as unfavourable a sign when it sets in after a 'stationary period' as when it occurs immediately after the period of initial lowering. As a rule, death is then not far distant, and the temperature may rise as before stated, to any point between 104° and 108°—the maximum being attained at the time of death, or very shortly afterwards. During this stage, too, the pulse and respiration are much more constant in their characters than they are in the earlier stages of the apoplectic attack. The pulse becomes small and very frequent—120 to 136 per minute. The respirations increase in frequency (48 to 64), and are also laboured, noisy, and often complicated with laryngo-tracheal râles. At the same time the extremities become dusky, especially on the paralysed side; while the face is red, swollen, and covered with an abundant clammy sweat. Finally, there is absolute muscular resolution in all parts of the body—even where more or less rigidity had previously existed—which continues till death closes the scene.

Temperature Variations indicative of Incipient Cerebral Softening (from Vascular Occlusions). Bourneville found that, with the exception of a few unusual cases, the 'period of initial lowering' of temperature commonly met with in cerebral hæmorrhage, is either absent or much less slightly marked where we have to do with incipient cerebral softening. During the first two hours after the occlusion of one of the middle cerebrals, for instance, the rectal temperature in such cases of incipient cerebral softening may range between 98° and 100°; while during the same period in cerebral hæmorrhage it is almost always below 97·2°. But after the first two hours in a case of softening, the temperature may suddenly rise to 102°, or even 104°, though it soon again descends to the normal standard, and afterwards exhibits altogether irregular oscillations—sometimes remaining the same for two days, and sometimes presenting evening or morning remissions of two degrees.

Among the exceptions to this order of events in cerebral softening we have to include most instances in which the pons Varolii is thus affected. In one of my recent patients, in whom after death I found a thrombosis of the basilar artery, an apoplectic condition was met with very closely resembling that which exists in some of the largest cerebral hæmorrhages. The patient was profoundly comatose, and the rectal temperature had sunk in two hours from the onset of the attack slightly below 95°, and it continued at this extremely low level for three hours and a half, or till the patient died. This I believe is the lowest temperature ever recorded in a case of incipient cerebral softening, and it is likewise the most rapidly fatal case of thrombosis of the basilar artery yet on record. The only diagnostic indication which could in any way serve to distinguish this case of vascular occlusion from a large hæmorrhage into the pons Varolii, was the fact that the lowering of temperature went on slowly, taking two hours to attain the extremely low figure above indicated; and also the fact that the pupils were not markedly contracted and were to some extent sensitive to light. Unfortunately, in other cases of this kind recorded by Hayem, no mention is made as to the state of the temperature, so that it remains for future observations to decide whether such a low temperature as that which I have met with would prove the rule in similar cases of complete occlusion, by thrombosis or embolism, of the basilar artery. It is, however, perfectly established that in the later stages of softening of the pons Varolii, the temperature very frequently rises before death to 108° or even 110°. Many years ago I saw a case of this kind in which the latter temperature was attained before death, and in this case a very considerable portion of the pons was subsequently found to be in a semi-diffluent condition. The pons Varolii is, however, an altogether exceptional region of the brain, owing to the fact that within its boundaries, or in immediately adjacent parts of the medulla, the chief vaso-motor centre is situated; and also because within these same parts, if there be not a centre or mechanism that regulates (or in some way affects) the metabolic processes taking place in the tissues, so as to produce at times marked depression and at others marked elevation of body-heat, there is, at least, an increased liability for lesions within this narrow area to implicate efferent fibres proceeding from such centres, situated, it may be, within the cerebral cortex (so-called 'Heat-Centres').

In what has been said before, therefore, as well as in what is to follow, we must not be understood to refer to the temperatures that occur in cases of softening in the pons Varolii.

In cerebral hæmorrhage, while the 'initial lowering' of temperature is generally much more marked than in cerebral softening (owing, doubtless, to the greater shock from laceration of brain tissue), it is found that if the temperature rises soon after the attack to a point beyond 102·5°, it rarely sinks again to the normal standard, except under the influence of a shock resulting from fresh hæmorrhage. The sudden rise to 102° or 104°, followed by a speedy fall, becomes, therefore, very suggestive of the existence of softening rather than hæmorrhage, unless there is reason to believe that the fall has been occasioned by a fresh accident within the cranium. Again, in the so-called 'stationary period,' the oscillations of temperature, according to Bourneville, are more regular and slighter in amount in cases of cerebral hæmorrhage than in those of softening. Such a period lasts for a variable time, in instances of cerebral softening, though where one of these cases is about to prove fatal, the temperature wave becomes characterized by an 'ascending period'—even here, however, in the majority of instances, the rise in temperature takes place more slowly than where we have to do with the terminal period of a case of cerebral hæmorrhage. Ramollissement may terminate fatally, in fact, when the rise of temperature has been comparatively small—not more than to 102° to 104°. Though in other exceptional instances, as where we have to do with extensive softening of the pons Varolii, it may rise before death to the very high point already indicated.

The variations in the pulse and respiration afford no certain indications which may aid us to distinguish between cerebral hæmorrhage and cerebral softening. And, even in regard to these temperature variations above recorded, much more extended observations are needed in order to show whether the indications which we have already obtained will prove constant for the great mass of cases of hæmorrhage and softening respectively. We want especially more detailed information concerning the temperature curves for lesions of both kinds in different encephalic regions. This is a scientific development of the subject which is now much to be desired, as our knowledge of the temperature variations in cerebral hæmorrhage and cerebral softening respectively must be regarded as being still in an extremely elementary stage.

SECONDARY COMA.

1. EPILEPTIC COMA.
2. EPILEPTIC COMA IN GENERAL PARALYSIS OF THE INSANE.
3. EPILEPTIC COMA IN CEREBRO-SPINAL SCLEROSIS.
4. COMA WITH MENINGITIS.
5. COMA WITH TUMOUR OR ABSCESS OF THE BRAIN.
6. COMA WITH URÆMIA.
7. COMA WITH DIABETES.
8. COMA WITH ACUTE YELLOW ATROPHY OF THE LIVER.
9. COMA WITH HYPERPYREXIA.

As a matter of clinical convenience it is desirable to separate the comas which we have hitherto been considering as 'primary,' from those to which we shall now briefly allude as 'secondary.' We freely admit that this is an artificial distinction, the coma is in each case an effect of antecedent conditions—all that we mean to imply by the distinction is, that in the one set of cases we have to do with comas originating more or less abruptly in the midst of states of ordinary health; while in the other we have comas supervening in the course of some well-marked illness, from which the patient has previously been suffering. The first variety is the only apparent exception to this rule, since a patient who is subject to epileptic fits may be going about in what to others seems to be a state of apparent health, when he is suddenly seized with an attack which speedily renders him comatose.

1. Epileptic Coma. Taken by itself the coma that follows an epileptic fit presents no distinguishing peculiarities. If nothing is known as to the antecedents of a person found in this condition, the chances in favour of a case being one of epileptic coma, decidedly increase with the youth of the patient, because in early life epilepsy is common, just as in later life apoplexy from cerebral hæmorrhage or softening is common. So that, having to do with an elderly person, even if we learn from surrounding friends that the patient has been subject to epileptic attacks, there is only a strong presumption raised in the first instance that his condition may be a sequence of such an attack. It may of course be otherwise. A patient who has been subject to epileptic fits is not thereby protected from the occurrence of ruptures of, or occlusions in, some of his cerebral vessels: far from it. We have, therefore, always to take into account this possibility; and should invariably satisfy ourselves as to the absence of all signs of unilateral paralysis, and otherwise exclude the likelihood of the existence in the case before us of one of the more serious causes of the comatose condition, before resting satisfied with a diagnosis of epileptic coma.

2. Coma in General Paralysis of the Insane.

3. Coma in Disseminated Cerebro-Spinal Sclerosis.

In each of these allied conditions, occurring mostly in young or middle-aged adults, we are apt to meet with sudden epileptiform attacks (often continuing with intermissions for many hours), or else with attacks more distinctly apoplectiform in character.

The distinguishing clinical peculiarity of these attacks is that the temperature of the patient almost invariably begins to rise from the first, so that in the space of two hours it may be 101°, and in four or five hours more (or sometimes within a shorter period) it may have reached 104°. Where the convulsions cease and the patient begins to regain consciousness, the temperature may, after a few hours, gradually sink to its normal level. In more severe cases in which the termination is to be unfavourable, however, the temperature may rise still higher, and death may take place in the course of a few days.

During such attacks, the pulse is generally much increased in frequency, and the convulsions when present are mostly unilateral. After death, in these cases, no recent lesion is to be met with to the occurrence of which the convulsions or coma can be ascribed. The sudden elevation of temperature is, therefore, a sign of great diagnostic significance for the discrimination of such functional or so-called 'congestive attacks' from apoplectic conditions due to cerebral hæmorrhage and cerebral softening on the one hand, or to uræmia on the other.

Apart, however, from this characteristic, there would be the evidence as to the previous condition of the patient suffering from one or other of these usually well-marked diseases.

It should be mentioned in this connection that a 'status epilepticus' occurring in an ordinary epileptic patient (that is, a condition in which many fits occur in quick succession, the one following the other without intervals of consciousness) is commonly associated with a rise of rectal temperature to 104° F. or even 105°. A similarly rapid recurrence of fits in the condition known as hystero-epilepsy is said, according to Charcot, not to occasion any such rise of temperature—the rectal temperature remaining either normal or exceeding 100° only on the rarest occasions.

4. Coma with Meningitis. At a certain stage of traumatic, of simple idiopathic, or of tubercular meningitis, a condition of stupor or of actual coma may supervene. These are ordinary incidents in the course of these very grave diseases, but they are not likely to supervene before the nature of the underlying affection is recognized.

5. Coma with Tumour or Abscess of the Brain. In the terminal stages of tumour as well as of abscess of the brain, in a certain number of cases, insensibility and coma may supervene. Here again, it is the underlying diseases themselves which have to be recognized, seeing that they will generally have been in existence for some time—and occasionally for long periods—before the onset of anything like a condition of coma.

6. Coma with Uræmia. The diagnosis of uræmic coma may at times present considerable difficulties, though in other cases it is made with comparative ease.

In diagnosing uræmic coma we must look partly to the characters of the attack itself, and partly to the presence of certain physical signs with which this state is usually associated—viz., a pale pasty complexion, with puffy eyelids, swollen ankles and albuminous urine.

Here, however, it must be recollected that should it be a person beyond five and thirty or forty years of age who presents these characteristics testifying to the existence of renal disease, possibilities other than those of uræmia are also opened up. This general condition in such a patient would warrant our assuming the possible existence of a degenerated condition of the cerebral arteries—capable of favouring either hæmorrhage or thrombosis. The establishment of the existence of Bright's disease, therefore, in a middle-aged person, should not by any means incline us to pronounce too decidedly in favour of uræmic coma, as against an apoplectic condition due to cerebral hæmorrhage or softening.

In order to form a definite and reliable opinion we must always look, in these as in other cases, not only to the nature of the attack itself, but also, when possible, to its mode of onset and premonitory symptoms.

An attack of uræmic coma may or may not begin with convulsions, following prodromata in which drowsiness, headache, and sickness are usually marked symptoms. The coma is not profound. Patients suffering from uræmic coma may be momentarily roused with comparative ease, though they quickly relapse into their previous condition when no longer spoken to or otherwise disturbed, as in cases of narcotic poisoning. Twitchings of the limbs and rigidities, transitory and often shifting in locality, are not unfrequently met with. The pupils are extremely sluggish, but may be of medium size, dilated, or even contracted. The stertor, when present, is peculiar. It is not, as Grainger Stewart puts it, "the deep snoring of hæmorrhagic apoplexy, but a sharper, more hissing sound, produced by the rush of expired air on the hard palate or teeth." There is, with these symptoms, an absence of the signs of unilateral paralysis, together with a well-marked peculiarity in the temperature curve. Bourneville pointed out that the temperature of the body begins to fall with the onset of uræmic coma, and continues to sink as long as this condition persists. Thus the temperature of the body may ultimately fall as low as 90° in fatal cases of this type. On the other hand, in the coma due to cerebral hæmorrhage or softening, the lowering of the temperature is slighter in amount, and rarely lasts longer than from twelve to twenty-four hours in cases not fatal within this period—often, indeed, the lowering persists only for two or three hours.

The premonitory symptoms of uræmic poisoning, especially when they are of chronic type and associated with the cirrhotic form of Bright's disease, are apt to be confounded with tumour cerebri, if the existence of the chronic renal disease should be overlooked. We may have in both more or less intense headache, with occasional or frequent sickness, mental dulness, occasionally convulsions, and also some defects of sight. In tumour of the brain, there will often be optic neuritis or even atrophy ; whilst in chronic uræmia, there will, for the most part, be either no appreciable opthalmoscopic changes or else those of albuminuric retinitis.

7. Coma with Diabetes.—In diabetic patients of any age, death in an uncertain percentage of cases is apt to occur in a rather abrupt manner, through the supervention of a remarkable dyspnœa, speedily followed by stupor, coma, and death.

Alarming symptoms of breathlessness may set in suddenly, leading to inspirations and expirations of extraordinary fulness and depth. The patient pants like a person out of breath from over exertion. After few or many hours the surface becomes cold, the hands and lips blue, the breathing still remaining frequent though shallower; the pulse becomes rapid and small, and insensibility supervenes, gradually deepening into coma. During the stage of coma, twitchings may occur in the limbs or different parts of the body, but there are no signs of a unilateral paralysis. No abnormal physical signs are to be discovered in the lungs or heart in any way accounting for the dyspnœa.

The supervention of an attack like this in a patient suffering from diabetes, is very characteristic and distinctive. As to the cause of these attacks we have no precise knowledge. The sudden supervention of symptoms of acetone poisoning, or the sudden occurrence of fat embolism of the lungs and other parts, are theories which have been advanced to explain the condition. It is well to recollect that these alarming symptoms sometimes become developed under the influence of fatigue and excitement. A patient, for instance, has been suffering from diabetes for some time, and leaves home, we will say, in order to be admitted after a comparatively short railway journey into a hospital; yet before the patient has been in the hospital two days, dyspnœa and coma may become established and prove rapidly fatal. This occurred not long since with a patient admitted into University College Hospital under my care; and several such cases have been recorded.

It should be borne in mind that diabetes is occasionally associated with actual disease of the brain, or, rather, that softening or hæmorrhage may occur in diabetic patients; the nature of the attack itself, therefore, must be carefully studied before the diagnosis of diabetic coma is arrived at.

8. Coma with Acute Yellow Atrophy of the Liver.—This is an extremely rare condition which supervenes suddenly at times in patients who are apparently suffering from a simple attack of what might be mere catarrhal jaundice. After the jaundice has lasted some days or weeks, delirium alone or delirium and convulsions, may suddenly set in—soon to be followed by deep coma, stertorous breathing, and dilated pupils.

The condition of such a patient is carefully given in the notes of the following case recorded by E. J. Jenkins (Brit. Med. Jrnl., Feb. 23, 1884, p. 357):—An intemperate man, aged 43, had been suffering from sickness and other symptoms for three days, when he became jaundiced. Two days after this was recognised, at 4 a.m., he had a fit of violent delirium, during which the limbs became quite stiff; the teeth were ground together, and a considerable quantity of dark matter, like treacle, was vomited. He was delirious all day, and was brought to St. Bartholomew's Hospital at 4 p.m. and admitted under the care of Dr. Church. When seen in the cab he was violent, throwing his arms about, and making a great noise. When seen in the ward at 5 p.m., the patient was lying on his back, markedly, but not deeply, jaundiced; the pupils were widely dilated but sensitive to light; the breathing was stertorous, cheeks were puffed out, the teeth clenched, and every now and then ground together; the gums and lips were remarkably parched and leathery, the tongue (so much as could be seen, for the mouth could not be opened) was dry and black; the limbs were rigid so that they could not be flexed. The skin was light yellow, moist and cold; no hæmorrhages had occurred. The pulse was 118, irregular, and easily compressible. The rectal temperature was 97·8°. The area of dulness due to the liver was much diminished in extent. 6.15 p.m.—The patient was now sweating profusely; the pupils were widely dilated, and insensitive to light. The breathing was stertorous, forty-eight in the minute; the pulse was 132, fairly strong, but irregular. Rectal temperature 97°. Frequent spasms of short duration occurred, and all the limbs were in a state of rigid tonic contraction. About four drachms of dark treacley fluid were vomited. 8.30.—Still comatose; breathing 30, more stertorous and prolonged; pulse 130; rectal temperature 97°. 9.30 p.m.—Pulse 113; temperature 97°. The patient's breathing gradually became quieter and weaker, and he died at 11.15 p.m. At the autopsy the liver presented a characteristic appearance; whilst the brain and its membranes showed nothing unnatural.

9. Coma with Hyperpyrexia.—The kind of symptoms apt to be presented with this species of Coma have been described under the head of sunstroke (p. 59). The clinical characteristics of the condition are essentially similar when it supervenes in the course of rheumatic fever, typhus fever, pneumonia, variola, typhoid, puerperal fever, or pyæmia—during all of which it may occasionally present

CAUSES OF ENCEPHALIC PARALYSES.

- Paralyses of Rapid
  - 1. *Traumatisms.*
    - *a.* Lacerations of brain substance, or compression by depressed bone.
    - *b.* Hæmorrhages ...
      - (1) Meningeal (large or small).
      - (2) Intra-Encehaplic (single or multiple).
    - *c.* Meningeal Inflammations (occurring as a speedy sequence)
    - The existence of the Traumatism itself is generally obvious.
    - But which of the effects or which combination exists, is generally what requires to be decided—especially where there is the certainty or even possibility that alcoholic intoxication coexists.
    - On other occasions, the question arises whether we have to do merely with the effects of a fall (which is known) or with brain disease—the onset of which occasioned the fall.
  - 2. *Hæmorrhages*
    - *a.* Meningeal ...
      - (1) In consequence of falls or blows upon the head.
      - (2) In children during paroxysms of spasmodic coughing.
      - (3) From rupture of aneurysm on one of larger meningeal
    - Evidence of Hydatids in other Organs.
    - General signs of Encephalic Tumour.
  - 8. *Cysticerci* ...
    - Existence of Intestinal Tape-worm (and ? auto-infection through vomiting.) Signs of disease of Cerebral Cortex (Convulsions, Dementia, etc.; commonly no hemiplegia). Signs of Tumour Cerebri (general). All ages, but more common after middle life.
  - 9. *Simple Cysts.* Causing general symptoms of Intra-Cranial growth (*Supra* 4, *b*, (3)).
- Paralyses of Very Slow Onset.
  - 10. *Disseminated Cerebro-Spinal Sclerosis.*
    - Males and females equally—generally below 40 years. No cephalagia. No optic neuritis (often). Evidence of a dissecting lesion in medulla or pons, plus signs of lateral and posterior spinal sclerosis.

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itself. When hyperpyrexia comes on towards the close of tetanus, as a result of severe traumatic lesions implicating the cervical region of the cord, in tubercular meningitis, in softening or hæmorrhage of the pons Varolii, or in other serious forms of brain disease about to prove fatal, the particular condition previously existing must profoundly influence the combination of symptoms presented—yet, in the main, the hyperpyrexia itself tends to produce such symptoms as have been previously described.

(B.) PATHOLOGICAL DIAGNOSIS AFTER OR IN THE ABSENCE OF AN APOPLECTIC STAGE.

At the commencement of our consideration of Paralyses of Encephalic Origin, there will be found (p. 23) some General Considerations concerning the several Conditions which Cause such Paralyses. There all details which are of importance from the point of view of diagnosis have been set forth. Looking to this fact, nothing remains to be done in the present section but to gather up the several diagnostic indications in the briefest and most salient manner possible, and in such a way that they may be most readily grasped and contrasted with one another.

In order to carry out these objects, and to facilitate reference, the reader will find the differential diagnosis of the several pathological conditions, worked out in tabular forms, with references to the previous pages in which the diagnostic relations of such pathological conditions have been stated more at length.

In the first place the student will find in Table I. a general scheme referring to all the organic causes of Encephalic Paralysis.

This general table now requires to be carefully analysed, with the view of showing how the pathological diagnosis is gradually arrived at, by processes of exclusion in its earlier stages, and by the careful balancing of all kinds of evidences when the problem has been narrowed down to its ultimate issues.

In regard to the ten causes of Paralyses of Encephalic Origin given in the preceding Table, it is generally not difficult to decide in regard to the first of them. The cases of Traumatism are generally self-evident, so far as this mere first stage in the diagnosis is concerned.

The careful consideration of the very slow onset of the paralysis in Disseminated Sclerosis, together with the characters mentioned in the table, will often also suffice for the diagnosis of this tenth cause on our list.

Congenital diseases or Atrophy of Brain (not included in the foregoing tables) are generally distinguishable at once by reason of the arrested growth of one or two limbs, coupled often with mental defects.

There remain for consideration two contrasted groups of pathological conditions, viz., Intra-Cranial Hæmorrhages and Softenings on the one hand, and Intra-Cranial Adventitious Products on the other hand. These groups are composed and distinguished from one another as follows :—

			General Characters of Group.
TABLE II. Non-traumatic Intra-cranial Lesions—excepting Disseminated Sclerosis.	Producing Paralyses of Rapid Onset.	1. Intra-Cranial Hæmorrhage. 2. Thrombosis. 3. Embolism.	Paralysis occurring abruptly, or, if not, attaining its maximum within 2-3 days. Often ushered in without previous head-symptoms of any kind. Frequently commencing with an apoplectiform stage. (*See* Table III.)
			General Characters of Group.
	Producing Paralyses of Slow and Gradual Onset.	1. Tumours. 2. Abscesses. 3. Hydatids. 4. Cysticerci. 5. Simple Cysts.	Headache, Vomiting, Vertigo, and Optic Neuritis. Convulsions (often unilateral, or even still more partial). Failure of Memory, and Mental Dulness with increased irritability of Temper. Some of these signs often precede for weeks or months paralyses of cranial nerves or limbs (generally of progressive character). Coma often preceding death. (*See* Table IV.)

In Table II. are set forth the broad distinguishing features of these two classes of pathological conditions. And in Tables III. and IV. the leading points for the diagnosis of the different conditions included under each class are set forth. These tables refer to questions of great difficulty, and in actual practice all the knowledge and judgment of the student or practitioner will require to be brought to bear upon such problems, if a correct diagnosis is to be arrived at. Sometimes, when the indications seem almost equally balanced in two directions, nothing more than a tentative or alternate pathological diagnosis is possible.

TABLE III. Paralyses of Rapid Onset.	1. Intra-Cranial Hæmorrhages. pp. 25-28 and 60-73.	No age excluded, but most frequent from 50th-70th years. Onset generally abrupt—possibly during some exertion or straining. Often with insensibility, and sometimes deep Coma. Lowering of rectal temperature within one hour of onset to 97°-94°; in 2-3 hours it may rise to 101°, not often higher except in cases about to prove fatal; subsequent oscillations slighter and more regular than in Embolism or Thrombosis. Often slight exacerbation of symptoms with febrile reaction in 3–7 days. Made more probable by evidence of albuminuria, arterial degeneration, and hypertrophy of left ventricle.
	2. Thrombosis (Arterial). pp. 29-32 and 60-73.	May occur at any age, with general or local predisposing causes (p. 30). Onset generally not abrupt, or with any profound loss of consciousness, but there may be pains on affected side of head and mental dulness or confusion; and there may have been premonitory head symptoms for days before. Either no lowering or no marked lowering of rectal temperature during the first two hours (often at 98·4), then may rise to 102°–104° for a short time—subsequently varying much on consecutive days, or morning and evening. Sometimes remission of symptoms and diminution of paralysis after 3-4 days. Made more probable in childhood, youth, or early adult life by existence, or convalescence from, some acute disease; or by recent childbirth. Later in life by evidence of arterial degeneration, combined with a feeble and intermittent pulse. [Thrombosis in basilar artery (p. 71) excepted.]
	3. Embolism. pp. 33, 34 and 60-73.	Occurs in childhood or before 40th year, in a person having valvular disease of the heart. Embolism most common in the Sylvian artery—especially the left (*see*, therefore, whether the nature of the attack would accord with disease in this cerebral region). Onset of symptoms abrupt, perhaps immediately following some exertion, or emotional disturbance—generally with only slight and temporary, or perhaps no, loss of consciousness; but there may be pains on the affected side of the head, and some amount of mental dulness or confusion. Temperature variations much the same as those met with in thrombosis. Sometimes remission of symptoms and diminution of paralysis after 3-4 days. Made all the more probable if there has been previous evidence pointing to the occurrence of embolism in any other part of the body. [Multiple embolisms (p. 33) must be excepted.]

Table IV. Paralyses of Slow and Gradual Onset.	4. Tumours, Simple Cysts, and Pachymeningitis. pp. 4, 42, 49.	May occur at all ages. Often following blows; often not. Existence of some constitutional condition, such as Scrofulosis or Syphilis. Presence of tumours in other parts of the body. Convulsions unilateral, or even more limited in range. 'Late rigidity' not common. (General symptoms indicative of Tumor Cerebri. *See* Table II.)
	5. Abscesses. p. 41.	*a.* After Traumatisms (commonly in 6-12 weeks.) Course at first insidious, symptoms increasing rapidly at last. (General Symptoms of Tumour.) *b.* With Suppurative Otitis. (General symptoms of tumour or of tumour with partial meningitis; insignificant at first; rapidly developing at last.) *c.* In association with Pyæmic Conditions, or Empyema. (Febrile state with vague head symptoms, more rarely those definitely indicative of Tumor Cerebri.)
	6. Hydatids. p. 46.	Known excessive association with dogs, especially in countries where hydatid disease is common. Evidence favouring presence of hydatids in other organs or parts of the body. Age of patient generally between 10 and 30 years. (General symptoms of tumour in Cerebrum, not in Cerebellum.)
	7. Cysticerci. p. 48.	Proof of existence of tape-worm in the intestine of the person affected or of its recent existence (and therefore possibly of auto-infection through sickness, etc.) Signs of disease of Cerebral Cortex (Convulsions, dementia, etc., motor paralysis slight or absent). All ages, but more frequent after thirtieth year. (General symptoms indicative of Tumor Cerebri.)

It only remains now to give some few indications by the aid of which the student or practitioner may, in some cases, be able to arrive at a more precise diagnosis as to the nature of a particular New Growth or Tumour deemed to exist in any given case. Such a differential diagnosis is only possible in the case of some tumours, in some cases. In other instances, only a mere surmise can be offered as to the actual nature of the growth presumed to exist.

TABLE V.		
Tumours of the Brain and its Meninges.	Growths. p. 35.	Growths in brain often multiple (simultaneous or successive), affecting cerebellum with especial frequency—also common in cerebral cortex. Slight exacerbations, with febrile symptoms, from time to time. Sometimes ultimate development of Meningitis.
	b. Syphilitic Growths (Gummata or pachymeningitis). p. 35.	Symptoms rarely before 20th year. More common in males than in females. (A specific history dating 4-10 years previously.) Often signs that the disease exists in the parietal regions (partial paralysis and convulsions); sometimes rather about the base of brain, and then there may be double unsymmetrical paralyses of cranial nerves.
	c. Cancerous Growths. p. 36.	Much less frequent than either of above. Most common after middle age—but youth or childhood not exempt. May be primary, or secondary to cancer elsewhere. Growth generally solitary (more frequent in cerebrum than in cerebellum). Possible exacerbations in course of clinical history, from hæmorrhage into growth or softening set up around it. Rapid progress of symptoms (generally proves fatal in 6 months—still, in some cases, not under 2-5 years). Possibly perforation of cranium by new growth.
	d. Gliomata. Sarcomata. Myxomata. pp. 38–40.	One or other common, generally solitary and slow growing. Often follow blows upon the head. More common during first half of life. After scrofulous growths they are those most frequently met with in childhood and early adult life. Sudden exacerbations of symptoms from intercurrent hæmorrhages into the substance of the tumour. General nutrition good till late stages of disease.
	e. Exostoses. p. 40.	An internal exostosis may correspond in site with some external exostosis or irregularity of cranial bone. Occasional development in site of previous blow. Progress often extremely slow. Existence of exostoses from other bones.
	f. Tumours of Pituitary Body. p. 40.	May cause temporal hemiopia; or, if developing more on one side than on the other, amblyopia or amaurosis of this side, together with incomplete hemiplegia of opposite side (of gradual and probably non-simultaneous development). Paralysis, complete or partial, of 3rd, 4th, and 6th nerves on side of amblyopia.
	g. Fibromata, Lipomata, Psammomata, Pappilomata, and Osteomata. p. 40.	Rare or insignificant growths, having no distinctive history or characters capable of being used for the foundation of a diagnosis.

REGIONAL DIAGNOSIS.

GENERAL CONSIDERATIONS RELATING TO THE REGIONAL DIAGNOSIS, IN PARALYSES OF ENCEPHALIC ORIGIN.

Regional or Localising Value of Special Symptoms that may be associated with the Paralysis.

In the present section of this work we do not pretend to deal with the localising value of the several symptoms in an exhaustive way, but only to set down such facts as may be of practical use, in aiding the student or practitioner to arrive at a correct regional diagnosis in the various cases of paralysing brain disease that may come before them.

Some of the signs and symptoms to which we shall have to refer possess no localising value ; the presence of others, however, affords most valuable information as to the precise region in which an injury or pathological lesion is situated.

Loss of consciousness (insensibility or coma) is a symptom belonging to the former category. It may occur as a consequence of lesions of the most varied nature and site within the cranium ; and whether it occurs or not in any given case, will depend much more upon the suddenness and extent of the lesion than upon its precise locality. Shock, vascular disturbance, and undue pressure within the cranium, are, perhaps, the most potent factors in its production.

Delirium, mania, melancholia and some other forms of insanity, on the other hand, have some though only a vague localising value. We may know that some parts of the cortex are diseased or in a state of perverted activity ; but the presence of one or other of such symptoms does not tell us what part of the cortex, of one or both hemispheres, is in a state of perverted activity in this or that particular case.

Other conditions, such as the existence of certain forms of unilateral spasm, or the presence of aphasia, either without or with very partial right sided paralysis, may, on the other hand, afford the most precise indications as to the region of brain affected.

In regard to aphasia, however, it is needful to say that what is said above will hold good only for the aphasic condition pure and simple; it will certainly not hold good if (as is only too frequently the case) this name be applied to almost any speech-defect that may present itself, without having proper regard to its precise nature. The loose use of this term must be corrected by a more accurate clinical discrimination of the various forms of speech defect. Otherwise the doctrines of cerebral localisation will be confused and retarded in their development.

The special signs and symptoms now to be referred to are as follows :—

I.—CONVULSIONS.
II.—DELIRIUM.
III.—MENTAL DEFECTS (Insanity, Imbecility, etc.).
IV.—CONJUGATE DEVIATION OF THE EYES AND HEAD.
V.—DISORDERS OF INTELLECTUAL EXPRESSION BY SPEECH AND WRITING (Aphemia, Agraphia, Aphasia, and Amnesia).
VI.—LOSS OF CONTROL OVER SPHINCTERS.
VII.—DIFFICULTIES IN DEGLUTITION.
VIII.—CEPHALAGIA AND VOMITING.
IX.—OPTIC NEURITIS AND OPTIC ATROPHY.
X.—AMAUROSIS, AMBLYOPIA, HEMIOPIA, AND DIPLOPIA.
XI.—PARALYSES ON CRANIAL NERVES.
XII.—HEMIANÆSTHESIA—CEREBRAL AND MŒSOCEPHALIC.
XIII.—DIFFERENCES OF TEMPERATURE ON THE TWO SIDES OF THE BODY—PARALYSED AND NON-PARALYSED.
XIV.—DISTRIBUTION OF MOTOR PARALYSIS IN FACE, LIMBS, AND TRUNK MUSCLES.
XV.—DIMINUTION OR EXALTATION OF REFLEXES.
XVI.—ASSOCIATED MOVEMENTS.
XVII.—RIGIDITIES (Contractures), EARLY AND LATE.
XVIII.—POST-HEMIPLEGIC DISORDERS OF MOVEMENT (Chorea, Athetosis, etc.).
XIX.—SECONDARY DEGENERATIONS.
XX.—CAUSATION OF CONTRACTURE, ANKLE CLONUS, AND EXAGGERATED KNEE-JERK.
XXI.—SECONDARY TROPHIC CHANGES IN JOINTS, NERVES AND MUSCLES.
XXII.—OTHER TROPHIC CHANGES IN CONNECTION WITH BRAIN LESIONS.

I. CONVULSIONS.

The actual cerebral processes by means of which convulsions are produced are at present almost entirely unknown. It is important to note that I say 'produced' and not 'induced.' There is a wide difference between the two meanings, the non-appreciation of which is apt to lead to many mistakes. For instance, because the irritation of certain parts of the cortex of the brain are now known to induce unilateral convulsions, many assume and speak as though the production of such convulsions could be adequately accounted for by 'discharging processes' in certain cells in the region of the cortex irritated; but such a process as this may be far from anything like an adequate explanation of the cerebral processes concerned in the actual production of an epileptic attack.

Irritation of the mucous membrane of the intestine in some children will lead to, or induce, the occurrence of convulsive attacks. It would be manifestly wrong, and nobody in such a case would think of saying, that the irritation of the intestine produced the attack—if the word is to be used in the sense above indicated. And yet irritation of the cells of the cortex may no more produce the attack (in this sense), than does the irritation of the intestinal mucous membrane produce the attack in the case of the child. In each case, the processes named may merely upset the balance of encephalic nervous functions, in such a way as to determine or permit the discharge of certain motor centres upon groups of pontine, bulbar, and spinal motor cells, both simultaneously and successively. But, if it be asked, what motor centres are they which discharge upon the bulbar and spinal ganglion cells, or what are the ultimate or penultimate nervous processes that determine such discharges, the writer is inclined to say that on this subject we know very little. The irritation of the cortex may lie almost as much outside these discharging processes and their immediately determining conditions, as the irritation of the intestine to which we have alluded. They are each of them more or less remote links in the chain of causation. It is true, that these two causes of convulsions may disturb the balance of encephalic functions in different ways. The one may disturb the balance from the motor side, the other may disturb it from the sensory side—in the one case we should have molecular movements descending to the pons Varolii, in the other we should have molecular movements ascending to the pons Varolii. In this part of the brain cerebral and cerebellar nerve processes are, probably, to some extent, in a condition of antagonism.

What has been said above, is applicable to the association of convulsions with lesions in many different parts of the brain ; for it is a fact that, convulsions are apt to occur with different lesions situated in many parts of the brain. In the majority of such cases, nearly all that can be said is, that such lesions induce convulsions, although they may only take a remote part in the actual mechanism of their production.

Even if we admit all this, two questions naturally present themselves :—(A). What are the parts of the brain in which the occurrence of lesions is most prone to be associated with convulsions ? (B). Is there anything in the form or nature of the convulsion which tells in favour of its incitation by a lesion in this or that locality ?

(A).—Convulsions are most prone to occur with lesions in the following localities :—

(a).—Lesions of the Cerebral Cortex, especially in the posterior frontal and in the parietal regions (Fig. 6).

(b).—Lesions causing effusion of blood into the Lateral Ventricles.

(c).—Lesions of the pons Varolii.

(d).—Lesions of the middle lobe of the Cerebellum.

(e).—Inflammation of the Meninges.

(f).—Lesions in, or in the region of distribution of, the posterior third of, the hinder segment of the Internal Capsule (Fig. 10).

Lesions in other parts of the brain may at times, and under conditions connected with the nature of the lesion, as well as with the age, general state, and predisposition of the patient, be associated with convulsions. This association is, however, less constant when other regions are affected, than when we have to do with lesions in one or other of the above-mentioned situations. Even when these regions are affected, however, convulsions do not occur always ; they present themselves only in a variable proportion of the cases.

(B).—Some few facts may be cited in reference to the form of the convulsions, respectively associated with lesions in the different regions above indicated.

(a).—With lesions of the *Cerebral Cortex comprised within the posterior frontal and the parietal regions* (Rolandic Area), we have to do with highly characteristic forms of disease, known as Epileptic Hemiplegia or (as it has been more recently termed) Jacksonian Epilepsy.

These convulsive attacks, due to disease of the convolutions bordering the fissure of Rolando, are mostly unilateral, though in some cases they become general. They are usually preceded by a distinct aura in the form of numbness and tingling felt in the part first affected. The spasm may, indeed, begin comparatively slowly, either in the hand, especially in the thumb and forefinger; in the face or tongue, or both these parts; or in the foot, and especially in the great toe.

The range of the spasm varies immensely in different cases. We may have all gradations between slight spasm limited to the thumb and index finger, and universal convulsion. Different attacks in the same patient may vary widely in this respect. Practically, in regard to range of spasm, as Hughlings Jackson points out, these attacks are divisible into three classes, viz.,

(1) Monospasms (of arm, of face, or of leg);

(2) Hemispasms of two degrees,—

(a) Convulsions of face, arm, and leg,

(b) Convulsions of face, arm, and leg, with the addition of turning of the eyes and head to the side convulsed (the analogue of cases of hemiplegia described by Vulpian and Prevost, in which, owing to destructive lesions, the eyes and head turn from the side paralysed).

(3) Lastly, in certain cases a further spread of the convulsion takes place, so that a part or the whole of the other side of the body is involved, and the convulsion thus becomes more or less universal.

When the spasm starts and spreads slowly it commonly remains limited, and there is then often no loss of consciousness. When it sets in more abruptly and spreads more quickly, at some stage, generally before the whole of the side is affected, consciousness is lost. The processes may be thus limited; or, consciousness being lost, the spasm may then spread, as above indicated, so as to involve the other side of the body. The duration of the attack is extremely variable; it ranges from a minute or two to an hour or more.

After unilateral convulsions of this type, whether partial or complete, paralysis or marked paresis of the limbs affected, may exist for some hours, or even for two or three days. Where the paresis is limited, it is almost sure to occur in the parts which are first and most convulsed. It varies in degree from mere awkwardness in finer movements, to absolute powerlessness of the hand and arm. The range may vary from mere powerlessness in the hand to a complete temporary hemiplegia. During this paralytic condition and also after it has been recovered from, for many days or it may be for weeks, there is a very distinct exaggeration of the knee-jerk and ankle clonus on the affected side. In some cases immediately after the convulsive fit they are absent for a minute or two. Sensibility is intact; but the affected limbs are often distinctly colder than those of the opposite side.

After right-sided fits of this kind beginning in the arm or in the side of the mouth, temporary and slight speech defects are occasionally met with—sometimes of an aphasic, and sometimes rather more of an amnesic character.

(b).—In unilateral lesions of the cerebrum leading to *effusion of blood into the Lateral Ventricles*, there is nothing very distinctive in the nature of the convulsions that often occur. We must be guided, therefore, in great part by the general assemblage of symptoms indicative of the existence of such a lesion. When the convulsions exist they are commonly bilateral, often of short duration, and frequently recurring. Sometimes there may be mere twitching on one side and marked convulsion on the other. These convulsions are held by Pitres to be due to irritation of fibres entering into the wall of the anterior half of the lateral ventricle—fibres which pass downwards from the Rolandic area to form part of the internal capsule.

(c).—In the convulsions, again, which occur at the onset, or during the progress, of some cases of *disease of the pons Varolii*, there is nothing distinctive in the nature of the attacks themselves. They are, however, commonly of a bilateral type. The diagnosis must depend upon the particular grouping of attendant signs and symptoms.

(d).—*Lesions of the middle lobe of the Cerebellum* are apt to be associated with convulsions of a tetanic character, often without loss of consciousness, in which the muscles of the back of the neck are in a state of spasm, drawing the head backwards, whilst the limbs, also, may be more or less rigid. Thus there may be some amount of opisthotonos, though it is commonly of an abortive type.

Attacks of this kind should always suggest the possibility of cerebellar disease, just as (though not with the same degree of certainty) attacks of Jacksonian epilepsy point to an initiation of the fits through structural or functional defect in the convolutions bordering the fissure of Rolando.

(e).—*Inflammation of the Meninges*, whether tubercular or nontubercular, whether idiopathic or traumatic, is very liable to be associated with convulsions.

Such convulsions are more common in association with tubercular meningitis than with either of the other forms, excepting syphilitic pachymeningitis.

The convulsions that occur in association with these morbid conditions may be of the most diverse kinds, simply because they may be induced in very various ways, by associated morbid processes differing much among themselves, acting upon the most diverse parts of the brain. They may be incited from this or that part of the cortex, owing to extension of the inflammatory process; or, in the case of tubercular meningitis, they may be due to the irritation caused by small tubercular growths, situated in one or other of the corpora striata, in the pons, or in the cerebellum.

(f).—*Lesions or functional perturbations in, or in the region of distribution of, the posterior third of the hinder segment of the Internal Capsule* (sensory division), ought to receive some mention here, even though the mention be, in part, somewhat dubious and uncertain in regard to precise locality.

The attacks to which attention is to be called, here, are those of so-called typical 'hystero-epilepsy,' upon which much light has been thrown during recent years by Charcot. Since many of the phenomena associated with this condition, are also met with in cases where actual structural lesions have existed in the posterior third of the hinder segment of the internal capsule, there is good reason for supposing that hystero-epilepsy is a condition having intimate relations with functional perturbations in this region of the brain.

The patients affected with this disease, which is exceedingly rare in its fully developed form in this country, though much more common in France, are commonly hemianæsthetic on the left side, though they may exhibit marked tenderness in the left ovarian region. Others present anæsthesia on both sides of the body, and double ovarian tenderness.

Fits may recur several times daily, or at much longer intervals. They often occur without apparent cause; though they may also be induced at will by pressure upon some particular part of the body, such as the sides of the thorax or the lumbar regions of the trunk.

A remarkable and complex aura occurs in the cases in which the attacks commence spontaneously, though it seems to be absent when the attack is induced by external manipulation. The characters of the aura are these :—It is comparatively slow and deliberate in its first stages, beginning with a painful sensation in the left ovary (lasting 3″-15″) ; followed immediately by a sensation of 'globus' starting in the epigastric region and associated with a sensation of swelling and stifling. Then follow palpitations of the heart and laryngismus; afterwards throbbing in the left temple, together with loud hissing and other sounds in the left ear. (The duration of these sensations may be 2″-3″ or more.)

The actual fit then begins, often with a loud cry, and is divisible into the following stages :—(1). The head is thrown violently backwards; the limbs and body become rigid; the respirations are infrequent and stertorous; the eyes open and shut alternately, the face is red, the pupils dilated. Then clonic contractions may follow on both sides of the body, equally or unequally ; and foam, or foam and blood, may issue from the mouth. (This epileptiform stage lasts 2″-3″.)

After an interval of two or three seconds the more specially hysteroid part of the attack begins, and is divisible into two stages—corresponding with the second and third stages of the attack as a whole.

(2).—The second period is characterised by violent movements. The pelvis is jerked forward several times in succession, the upper half of the body is thrown backwards and forwards with extreme rapidity ; after which the convulsions extend over the whole body without having any precise direction or being of any clearly definable nature; several persons are often needed to hold the patient down at this stage. (This period "phase de grands mouvements," terminates abruptly after about 3″-5″.)

(3).—With the commencement of the third stage (the stage of emotional attitudes) the patient may lie flat on her back with lower limbs stretched out straight and the upper at right angles with the trunk. After a few seconds the patient assumes the expression of face, together with the attitudes and gestures that characterise a succession of emotions, intense and varied in their nature.

(a) The patient sits up in bed and seems to menace or threaten some one for a moment or two ; then almost immediately (b) the whole expression of the face changes for one of abject, cowering fear, which lasts for an equally short period. This is followed by (c) a look of calm and saintly happiness ; then comes an expression of intense joy, (d) she seems to see some one whom she loves, she beckons, opens her arms and appears to embrace some one passionately. Again (e) fear takes possession of the patient, she screams at imaginary rats or some other objects which fill her with terror and disgust; then the fear subsides, the patient hears strains of music or voices singing, and she herself (f) may for a moment or two beat time or hum a tune. Then comes the last stage (g) in which the patient sits up, hides her face in her hands and weeps, reproaching her parents with having been the cause of her misery; this lasts two or three minutes, then all is over, and the patient is well, but for the fact that some hallucinations may persist for a time.

The account given above of the third stage of the attack corresponds with what has been observed over and over again in one of Charcot's patients (Louise A. G.). The reality of each emotional phase, and of the imaginary circumstances upon which they are based, were capable of verification in her case as in others. For it is a characteristic feature of these attacks, that they may be cut short at almost any stage, by exerting very firm pressure over the ovary on the affected side. The instant this firm pressure is made in the ovarian region two results follow:—first, the patient's mouth opens and the tongue is spasmodically extruded; and, secondly, the convulsions cease. When this manœuvre is executed during any period of the third stage, the patient can always describe the hallucination with which she was possessed at the particular instant, and it has been found that the hallucinations corresponding with the several stages are always constant for each patient—that is, that the same attitude always corresponds with the same hallucination. Some hallucinations begin during the second stage of the attack, though the first, or epileptiform, stage seems to be free from them—as has been ascertained by cutting the attack short at this period. After the attacks, sometimes trismus or rigidity of limbs may show themselves and persist for a longer or shorter period—hours or days.

As before indicated, fully developed attacks of the kind above described are rarely or ever met with in this country. The excitable temperament met with in some Frenchwomen, seems to be needful for their production.

II. DELIRIUM.

The existence of delirium in any given case of brain disease has only a localising value of a general kind. It enables us to feel sure that the cortical grey matter of the cerebral hemispheres is in a state of perverted activity. But it does not enable us to go much further. We cannot from the existence of delirium in any of its varieties, affirm that the perverted activity is in this or that part of the cortex, or even in this or that hemisphere, rather than in both.

Nay, more, seeing that delirium, especially in children, is excited with comparative ease by many causes other than organic disease of the brain, we must satisfy ourselves as to the absence of such causes (such as ordinary febrile diseases, high temperature howsoever induced, the action of poisons, etc.), before we should be warranted in supposing that an existing delirium stood in relation to organic intra-cranial disease of any kind.

Of organic diseases, it is most frequently in relation with meningitis or meningo-encephalitis. It may, however, occur also in relation with tumours, cysticerci, or some other organic disease of the brain which, at any period of its progress entails marked active hyperæmia of the cortex cerebri.

III. MENTAL DEFECTS.

Under this heading 'mental defects,' must be included symptoms of wide range notably different from one another. At one end of the series, we have such grave defects as are included under the various forms of insanity; and at the other extremity, the mere slowness of apprehension, or defects of memory, which are apt to be associated with grave organic disease of the brain. Sometimes such defects may be more apparent than real; or, in other cases, they may be defects dependent upon some limited sensorial or perceptional failure such as occur in so-called 'word-blindness' or 'word-deafness.' This latter class of cases will be referred to in another section (V.).

With such grave mental defects as are found in the various forms of Insanity we are not now specially concerned. Their existence affords us at present no other localising indication than that the functional integrity of the cortical grey matter is at fault—in different ways in different cases.

Looking at the question from the other side, it may be useful to make certain general statements in reference to the association of mental defects with such common brain lesions as hæmorrhage, softening, or tumour, in different localities.

Mental defects are apt to occur, with special frequency, where both hemispheres of the brain are simultaneously affected with organic disease.

Mental degradation, again, is prone to show itself during the later stages of certain cases of tumour of the brain, especially of those belonging to two categories :—First, cases in which the tumour itself becomes so large as to produce a considerable amount of general intra-cranial pressure ; and, secondly, cases in which, though not itself large, the growth (as in cases of tumour of the middle lobe of the cerebellum) by its pressure upon veins or sinuses, and by closure of the Sylvian aqueduct, leads to chronic hydrocephalus, and thus again by another mechanism to a great increase of general pressure within the cranium.

Where one hemisphere only of the brain is affected, and where the disease is not of such a kind as to produce an appreciable increase of general pressure (that is where the evidence of such a condition is wanting), and yet distinct mental defects exist, can anything be said as to the region of the cerebral hemisphere most likely to be affected? In other words, in what part of the cerebral hemisphere is the presence of disease most likely to be associated with distinct mental defects?

I long ago came to the conclusion, and expressed the opinion, that lesions in the posterior lobes, much more than in other regions of the hemispheres, are prone to be associated with mental defects of a marked kind. Rosenthal also has arrived at the same opinion. He says :—" In the case of new growths in the posterior lobes the psychical disturbances are incomparably more frequent than in that of tumours of the anterior or middle lobes." Hughlings Jackson strongly supports the same view ; he thinks, further, that these defects are more marked when the lesion is in the right than when it is in the left occipital lobe.

On the other hand, in opposition to these views, there is the fact that lesions in the occipital lobes are at times latent, and do not lead to any readily appreciable mental change. (See section on 'Signs and Symptoms of Lesions in the Occipital Region,' where this difficulty is specially referred to.)

Again, we shall have occasion to point out in sec. V. that the various forms of Amnesia are especially apt to occur in association with lesions situated in the convolutions immediately surrounding the posterior extremity of the Sylvian fissure. In 1880 in reference to this subject I first formulated the general principle ('Brain as an Organ of Mind,' p. 687) that " the tendency to mental impairment with Aphasia, and the degree of such impairment, will, other things being equal, increase as lesions of the left hemisphere recede in site from the third frontal convolution, and approach the occipital lobe."

Lesions of the anterior lobe often occur without producing any very definite mental defects. Still, at times, distinct alterations in the general character and disposition of the individual have been associated with disease of the anterior lobe of the brain. Ferrier believes that the power of attentive and intelligent observation is distinctly weakened with lesions in this situation. Crichton Browne also considers that such early symptoms of General Paralysis of the Insane as "general restlessness and unsteadiness of mind, with impairment of attention, alternating with apathy and drowsiness," coincide more especially with the morbid changes which are particularly prone to occur at this early stage of the disease in the cortex of the frontal lobes. This, however, would mostly be the result of bilateral lesions, and we might expect that such lesions would produce more marked effects than when we have to do merely with lesions of one frontal lobe. These latter cases have not unfrequently run a latent course throughout, owing to the absence of all definite or readily recognisable results. This subject will be more fully discussed hereafter.

IV. CONJUGATE DEVIATION OF THE EYES AND HEAD.

This symptom has already been referred to as a frequent accompaniment of the apoplectic state (p. 61), and one whose occurrence has great diagnostic significance. We have there pointed out the extent to which it is a sign having a general localising value, that is, as pointing to an implication of one or other hemisphere, according to the side to which deviation occurs. We have shown, moreover, that the indications which it affords, are the direct reverse of one another in simple paralytic, and in unilateral irritative or convulsive, affections respectively. Thus, we may have in the early stages of an illness due to certain lesions in the left hemisphere, right unilateral convulsions with deviation of the head and eyes to the right side (according to the rule in such cases); but if, in the course of some hours or days, the previous irritative lesion should lapse into a destructive lesion occupying the same site, then we should have right hemiplegia replacing the right-sided spasms, and with it a deviation of the head and eyes to the left, or side of lesion. That is to say, when an irritative lesion exists in certain parts of the left hemisphere it causes lateral deviation away from the site of lesion; but whenever the irritative passes into a destructive lesion, then the direction in which the head and eyes are turned changes sides, and the deviation is seen to be towards the side of lesion, or away from the paralysed side.

It was also pointed out that the above rules had to be reversed when the irritative or paralytic lesions were situated in either half of the pons Varolii.

It remains now to inquire, whether the occurrence of this sign is specially related to lesions in any particular parts of the cerebral hemispheres.

Ferrier ('Localisation of Cerebral Disease,' p. 58) calls attention to this sign, as it exists in association with paralysing lesions, under the name of 'Unilateral Oculo-motor Monoplegia.' Concerning the related monospasm he says:—"At the base of the first frontal, and extending partly into the second frontal convolution in the brain of the monkey there is an area (12, Fig. 6) irritation of which causes elevation of eyelids, dilatation of the pupils, conjugate deviation of the eyes and turning of the head to the opposite side. I have

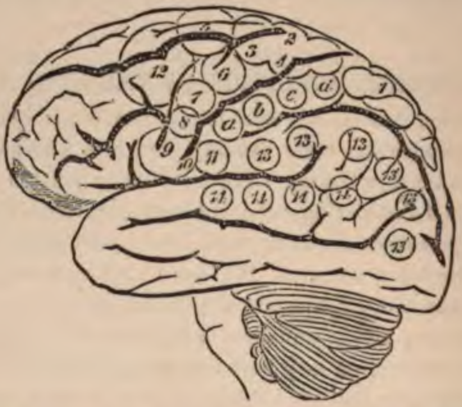

FIG. 6. HUMAN BRAIN SHOWING THE SITUATIONS OF ITS SO-CALLED MOTOR, AND SENSORY AREAS [after Ferrier].

12, indicates the region the excitation of which produces conjugate deviation of the head and eyes to the right, according to Ferrier. (The explanation of the other numerals will be found where this figure is repeated as Fig. 12.)

placed these various reactions in the order in which they occur with slight and longer continued stimulation respectively. In the faintest form of stimulation, elevation of the eyelids is the only effect observable. Whether individual centres, incapable of sharp demarcation from each other, for each of the movements indicated exist here, has not been determined experimentally."

Ferrier believes that this is the only region of the cortex, the irritation or the destruction of which gives rise, in opposite directions respectively, to conjugate deviation of the head and eyes. He calls attention, moreover, to several recorded cases in which this sign has existed alone, or at all events without any other motor trouble. One of them, the case of Dr. Carroll, certainly points to the possible existence of a lesion somewhere in the region indicated by Ferrier; but the only one of them in which there was an autopsy, viz., that of Chouppe, affords doubtful evidence, since there were at least two lesions, and the interpretation given to the case by Ferrier, is quite open to question, and has, indeed, been criticised by Grasset, who is disposed to think that lesions in quite a different region of the cerebral cortex are most potent for the production of this sign.

Another region of the cortex was also found by Ferrier to give rise, on irritation, to deviation of the eyes and head to the opposite side, viz., the angular gyrus and the upper temporal gyrus (Fig. 6, 13, 13′, 14) (see 'Functions of the Brain,' pp. 171, 164). But these motor phenomena, thus produced, Ferrier regards as merely reflex movements consequent on the excitation of subjective visual or auditory sensations respectively. In reference to the effects of stimulation of the upper temporal convolution, regarded by Ferrier as the 'auditory centre,' he says:—"On repetition of the experiment several times, though the pricking of the ear and the turning of the head and eyes in the direction of the sound constantly occurred, the look of surprise and dilatation of the pupils ceased to be manifested. . . . It is not in my opinion possible to determine experimentally whether in this case the movements are called forth through the agency of the lower centres only, or whether they are dependent on the associated action of a special centre in the motor region of the brain." Consequently, neither here nor in his later work does Ferrier seem at all disposed to believe that lesions of this region of the cortex tend to produce such durable conjugate deviation of the head and eyes as we are accustomed to meet with clinically.

Grasset, however, records two cases which, in his opinion, tend to show that the convolutions capping the posterior extremity of the Sylvian fissure are the very parts of the cortex whose lesions are specially apt to be associated with conjugate deviation of the eyes and head. He finds some confirmation for this view, also, in the results of an analysis of previously recorded cases in which this sign has been present, and in a recent work ('Des Localisations dans les Maladies Cérébrales,' 3^{me} Éd., 1880, pl. 111) he gives figures representing the site of lesion in sixteen of such cases, in all of which Ferrier's region (12) appeared to be intact, although one or other of the convolutions about the posterior extremity of the Sylvian fissure was (either alone, or with other adjacent parts) the seat of lesion. He refers to ten cases, moreover, in which the two regions in question were affected simultaneously; but could discover none in which conjugate deviation of the eyes and head was present with a cortical lesion implicating only the bases of the second and first frontal convolutions.

According to Grasset, therefore, when conjugate deviation is to be attributed to a cortical lesion, the morbid change is most frequently situated in the convolutions capping the posterior extremity of the fissure of Sylvius (centres 13, 13′, and 14 of Ferrier). He also believes, however, that this sign may be caused by damage to the bundle of fibres which passes to this region, by way of the posterior part of the internal capsule, from the peduncle, and he tabulates fourteen cases of this kind.

Thus the question stands at present. The experimental evidence points, as Ferrier thinks, towards the bases of the second and first frontal convolutions; whilst the clinico-pathological evidence points, as Grasset indicates, rather towards the convolutions surrounding the posterior end of the fissure of Sylvius, as the cortical region in which the occurrence of lesions tends to produce conjugate deviation of the eyes and head.

Future observers must strive to throw some additional light upon this matter. It does not seem to me that any of Ferrier's criticisms tend to invalidate the conclusions of Grasset. Both Ferrier and Grasset may, indeed, be correct in their localisations; the one may point to the actual efferent centres, and the other to the sensory centres through which these efferent centres are commonly roused into activity, for the production of conjugate deviation of the eyes and head. Both region 12, as well as regions 13 and 14, may, therefore, be sites in which the occurrence of lesions tends to produce conjugate deviation of the eyes and head. Subsequently, we shall find that Aphasia is similarly related to lesions in the third frontal convolution in some cases, and in others to lesions in the convolutions around the posterior extremity of the Sylvian fissure.

V. DISORDERS OF INTELLECTUAL EXPRESSION BY SPEECH AND WRITING (APHEMIA, AGRAPHIA, APHASIA, AND AMNESIA).

These symptoms strictly speaking might well have been referred to under a previous section (III.), as they are all of them, in one or other sense, mental defects. They are, however, mental defects of a very special kind, intimately related to one another, yet complex in their several functional and structural relations, so that, for practical purposes and for the sake of precision, it is unquestionably much better that they should be dealt with in a separate section.

Reference will here be made to acquired Defects of Speech, that is, to defects of this type which have supervened after speech has been attained, and as a result of brain disease occurring in this or that locality.

Still, before doing so, it seems desirable that brief reference should be made to defects of this kind which result either from congenital disease, or from disease at some date prior to the manifestation of articulate speech. The most important of these congenital defects is deafness, which of itself inevitably entails mutism—the individuals thus afflicted being known as 'deaf-mutes.' It must be borne in mind, however, that this mutism or dumbness may also be brought about by absolute deafness occurring from any cause after birth, but before the child has begun to talk; or even after it has learned to talk, up to the fifth or the seventh year. In cases of the latter type, the child soon (when left without the customary guidance in articulatory acts derived through the sense of hearing) forgets how to speak and becomes dumb.

In addition to this class of cases, there are also those of congenital idiotcy without deafness, in which the child never learns to talk. There are also other cases, allied to the last, in which, owing to some intra-cranial lesion occurring either before, during, or soon after birth, the child's mental condition is greatly degraded as well as his motor power. In some of the less severe examples of this type which have come under my observation, speech has been merely deferred—perhaps till the fourth, fifth, or even the sixth year—and may after a time become established in a natural manner.

Among acquired Defects of Speech we may have to do with troubles of various degrees and kinds, supervening at any period between infancy and old age, of a temporary or permanent character. The great variations in the extent and nature of these defects, is due to the fact that the impeding condition or lesion may act (1) upon parts of the brain concerned with the genesis of thought and of the will to speak, or (2) upon some part of the nervous channels or centres concerned merely with the actuation of speech. Thus it happens that, in one set of cases, acquired defects of speech may be associated with marked alterations in the intelligence or mental condition of the patient; whilst, in the other, they may be represented by inability to express thought by spoken words, or else by mere defective articulation.

Articulation, as a mere motor act, may be interfered with in diverse modes. Where speech-movements are inco-ordinate, we have such common defects as stuttering or stammering; or else those less marked perversions which are met with in some cases of chorea. Again, where the movements concerned in speech are more simply defective, we have that indistinctness of articulation or blurred utterance which, in various degrees, is so commonly associated with different forms of paralysis due to cerebral disease. To this kind of defect the name **Aphemia** is commonly applied. It presents itself under many various conditions, and with very different degrees of completeness. It often shows itself, in its most extreme form, in glosso-laryngeal paralysis, or in other varieties of disease of the medulla oblongata. It may likewise occur in its lighter forms, as mere blurred or difficult articulation, from disease in the same locality, with or without the coexistence of lesions in other parts of the brain. This is the case, for instance, in general paralysis of the insane, and in disseminated cerebro-spinal paralysis. Again, slight aphemia is apt to occur in association with hemiplegia (especially when recent) caused by different kinds of lesions, in various parts of the brain between the medulla and the cerebral cortex. As a rule, it is both more marked and more persistent in hemiplegia due to disease in the pons Varolii; while with lesions higher up, it is apt to be slight and more transitory—especially where such lesions exist on the right side of the brain.

Lastly, it should be mentioned that in a very remarkable case observed by Dr. T. Barlow symmetrically occurring lesions in the third frontal convolution of each side, were associated with complete loss of speech. Although in such a case as this the aphemic disability reaches its maximum, and articulate utterance is abolished, yet such a defect is more likely to be spoken of as one of complete aphasia—for reasons which will subsequently appear.

Aphasia, in the strict and proper acceptation of the term, is that kind of defect of speech in which the patient is found to be unable to utter any proposition or sentence, though his occasional distinct pronunciation of some one or two words (whether appropriate or not), shows that his speechless condition is not due to a mere difficulty in the execution of the muscular processes needful for articulation. Moreover, the patient's intelligent manner and gestures may plainly show that he understands what is said, and is capable of thinking, even though he is quite unable to give expression to his thoughts either by speech or writing.

The patient's power of writing is necessarily interfered with when aphasia, as is so often the case, coexists with right hemiplegia. Many such patients, however, learn to write with the left hand to a variable extent; though others continue powerless in this respect. The variations as regards the power of writing in aphasics are, in fact, almost as marked as the variations in their power of speaking; though these two sides of the combined defect are by no means always equal in degree in the same individual. Thus, sometimes a man who is quite unable to express himself in spoken words, may be able to write a letter with comparatively few mistakes; whilst, on the other hand, the performance of another aphasic patient, without a copy before him, may be limited to writing his own name. More rarely it is found that, an aphasic patient (though not from want of manual power) is unable to write even a single letter—in attempting to do so he makes mere unmeaning strokes. This is the condition to which the term **Agraphia** has been applied. It has, however, often been also applied to cases of Aphasia in which the disability as regards writing happens to be decidedly in excess of the speech defect. It should be distinctly borne in mind, that agraphic defects are always met with as part and parcel of the aphasic condition; and that, for the most part, the name 'Agraphia' has been applied merely to those instances of the aphasic state in which defects of writing happen to have been extreme. They are simply cases where the characters on the reverse side of the shield happen to have been most marked, and which have, therefore, been named accordingly.

It is important always to discriminate the various defects of speech above referred to from another set of defects, commonly grouped as cases of **Amnesia.** This latter is, in reality, a generic term under which are included many varieties of speech defect, always requiring to be carefully distinguished from Aphasia, Agraphia and Aphemia, as above described—even though such varieties may coexist, as is not unfrequently the case, with one or other of the kinds of defect above referred to. In its commonest form, the essence of an Amnesic defect lies in the fact that the patient very frequently substitutes wrong words or names in the place of those he wishes to employ. In a bad case of this kind, the patient may be quite unable to arrange words into a sentence capable of conveying a definite meaning. His speech is thereby rendered more or less unintelligible. In the most extreme forms of Amnesia the patient may make use, not of words at all, but of a mere string of unmeaning articulate sounds.

More special types of amnesic defect have been described under the names of 'Word-Blindness' and 'Word-Deafness' respectively. The nature of these defects, and also of some others belonging to the amnesic class, which are equally special, will further on be more distinctly defined.

As already indicated Amnesia often coexists with one or other of the speech defects previously referred to; and it may show itself just as plainly when the patient writes as when he speaks.

The following are the modes in which these defects most frequently group themselves :—

(1).—Loss of power (more or less) of Speaking and of Writing (typical Aphasia).

(2).—Loss or impairment of power of Speaking, but power of writing preserved (Aphemia).

(3).—Loss or impairment of power of Writing, but power of speaking preserved (Agraphia).

(4).—Aphasic loss or impairment in power of Speaking, with amnesic defects in Writing.

(5).—Aphasic loss of power of Writing, with amnesic defects in Speaking.

(6).—Amnesic defects alone in Speech, in Writing, or in both modes of expression. (Under this head are included many different kinds of defect.)

These several defects are dependent upon lesions in very different parts of the brain, therefore it is of the highest importance from the point of view of cerebral localisation not to confound such conditions with one another. It is especially important that the term Aphasia should not be so loosely employed as it often is by both students and practitioners. On the one hand, it must not be confounded with mere difficulties in utterance (Aphemia) which are so common in different forms of hemiplegia; and, on the other, it is equally important that the term Aphasia should not be applied to clinical conditions belonging rather to the class Amnesia. Mistakes of this kind have often been made, and when the patients in question happen to have died and lesions have been found altogether away from the region of the third frontal convolution, such cases have been and are still apt to be quoted by sceptics with the view of showing that all attempts to localise brain functions are more or less impossible or hopeless. Yet, in cases of this kind, it is often the clinical diagnosis only which is at fault, so that, for instance, a confusion has been made between Aphasia and Amnesia, or Aphasia and Aphemia—that is between clinical conditions which may be caused by lesions in very different parts of the brain.

On account of the importance and comparative complexity of the subject, it is desirable, before saying anything further concerning the classification of these speech defects and their relations with lesions in different parts of the cerebral cortex, that some account should be given of the exact order of cerebral processes involved in Speaking, Writing, and Thinking, respectively. Some readers may not require any such information, and, that being the case, it will be easy for them to pass over the following account, which is, for the most part, an abstract of views published by the writer[*] on different occasions during the last sixteen years. To some, an explanation of this kind may be needful, for an adequate comprehension of this part of the symptomatology of brain disease.

SPEAKING, READING, WRITING, AND THINKING: BEING SOME ACCOUNT OF THE NATURE AND ORDER OF THE CEREBRAL PROCESSES INVOLVED IN THESE ACTS.

Speaking.—The young infant first begins to distinguish natural objects from one another by differences in shape, colour, touch, odour, etc., which these may present to its different senses; it is then taught (slowly and with difficulty) to associate some object possessing certain combined attributes by which it is remembered, with a certain articulate sound which has been often repeated whilst the object has been pointed at, till by dint of continual repetition this sound (or word) becomes identified with the various attributes of the object. When heard, this sound invariably recalls to memory the object, of which it may now be said to form a kind of additional attribute—just as the sight or touch of the same object will, in turn, call up the memory of the sound employed as its designation. At first these articulate sounds (or spoken words) are only connected with external objects, though soon certain adjectives, signifying approval or disapproval, are added as qualifying sounds. . . . . . This is the first stage passed through in the acquirement of a language—it is the mere learning to associate particular sounds with particular mental impressions, which association becomes so strong at last as to be almost inseparable, the thing unfailingly recalling to memory the sound, and the articulate sound as surely conjuring up a more or less vivid idea of the thing . . . . . it would seem pretty obvious that so far as the infant thinks by means of language, it does so by means of the remembered sounds of words—these are its linguistic symbols of thought, which must, however, be mixed up inextricably in its mind with other sense impressions, and more especially with those of sight.

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[*] 'The Physiology of Thinking', *Fortnightly Review*, Jany., 1869; 'On the Various Forms of Loss of Speech in Cerebral Disease', *Brit. & For. Med. Chir. Rev.*,' Jany. and Mar., 1869; 'On the Muscular Sense', *Brit. Med. Jrnl.*, April, 1869; and in 'The Brain as an Organ of Mind', 3rd Ed., pp. 601-690.

The next step is the development or acquirement by the individual child, of the power of articulating for himself the sounds which have hitherto been increasingly employed as mental symbols. The potentiality of attaining to such a power the child receives, in the main, as an inheritance from so many antecedent generations of men, that its actual manifestation—the acquisition, that is, of the power of Speaking—can only be regarded as a motor achievement of an order similar to some of those which may be included among the Instinctive Acts: the similarity being not so much with the instinctive acts that animals are born with the capacity of performing, but rather with those which manifest themselves a little later in life, and which (from their more gradual acquirement) might be thought not to be Instinctive Acts at all ('The Brain as an Organ of Mind,' p. 561).

A process of 'learning' to speak intervenes, in part, in the former case; but this is whilst the inherited structures are undergoing development in the child's nervous system.

At first the child's articulatory capacity is confined to mimicking—that is to say, it repeats such words only as have just been spoken to it; but after a time, when the act of emitting one or other of these sounds has become perfectly easy by constant repetition, the child gives utterance to them of its own accord, on the mere sight of the objects with which the sounds were originally associated in its mind. After a time, Thought and Language become inseparably associated, so that, during thought-processes, words are voluntarily or automatically recalled by the renewal of previous nerve actions in the Auditory Perceptive Centres; and such nerve processes are followed (though, as we shall subsequently see, not till another set of guiding impressions have been revived, of which for the moment it will be simpler to say nothing) by those complex combinations of muscular actions concerned in the articulation of the several words as they arise in thought.

Reading and Writing.—Reading and writing are accomplishments superadded to those of articulate speech. The child has already learned to associate certain objects, or particular states of consciousness, with definite Sounds (or Names); he has further gained the power of articulating these names for himself: so that when he begins to learn to Read, he gradually builds up a still further 'association,' by which certain written or printed hieroglyphics (representing letters in definite combinations) are linked to the already known states of consciousness (Perceptions, Ideas, etc.) and their sound representatives. The previous combinations are, therefore, supplemented by being correlated with new visual symbols; and it seems certain that in the act of Reading, the words, which are primarily perceived in the Visual Centre, would almost simultaneously recall corresponding sounds in the Auditory Centre (as part of the perceptive process itself). From the Auditory Centre the stimuli, inciting to the articulation of the corresponding words, would then pass to the Motor Centres in precisely the same manner (that is, with the same intermediate process, subsequently to be referred to) as in the case of ordinary Speech. (Reading aloud requires therefore, amongst other things, that the Commissural Fibres connecting the Visual with the Auditory Perceptive Centres should be intact.)

It almost invariably happens that the power of Writing is acquired after the individual has been taught to Speak and to Read more or less perfectly. During this course of instruction the pupil learns to associate the visual perceptions of the separate letters of words with certain muscular movements of the hands and fingers necessary to enable him to produce the written letters for himself, and afterwards to join them together so as to represent words. Here again, however, there is interposed an intermediate guiding process, just as in the case of speech, the nature of which we shall presently fully consider. The muscular movements ultimately learned are in all probability much more intimately associated with sight perceptions than with sound perceptions; though of course the Word as a revived sound-perception may be said to exist also during the act of Writing. (Writing from dictation, for instance, cannot be accomplished when the Commissural Fibres between the Auditory and the Visual Perceptive Centres are destroyed in the left hemisphere—that is, when the auditory stimulus is unable to evoke the inciting or controlling activity of the Visual Centre, for the production of Writing movements.)

Perception and Perceptive Centres.—James Mill ('Analysis of the Human Mind,' 1829, p. 68) says:—"Ideas, also, which have been so often conjoined that whenever one exists in the mind the other exists along with it, seem to run into one another, to coalesce, as it were, and out of many to form one idea, which idea, however, in reality complex, appears to be no less simple than any one of those of which it is compounded. . . . . . The word 'gold,' for example, or the word 'iron,' appears to express as simple an idea as the word 'colour' or the word 'sound.' Yet it is immediately seen that the idea of each of those metals is made up of the separate ideas of several sensations: colour, hardness, extension, weight. Those ideas, however, present themselves in such intimate union, that they are constantly spoken of as one, not many."

Ideas fuse themselves in this manner into clusters, or complex ideas, because, being only repetitions or weak copies of sensations, they are reproduced in the same order as the sensations. And the sensations in question habitually occur in clusters because the 'external objects' to which they correspond usually impress the organism simultaneously through different senses. If, in a dark room, my hand comes upon an orange or a book, either of these sensations of touch will immediately fuse with nascent ideas of other possible sensations from the same object (whatever it may be) so that this object is perceived as a present external reality. Thus, in a Perception, we have a present sensation linking itself indissolubly, by association, with a complex idea derived from our past experiences with similar objects.

Looked at from the physiological side the interpretation of such a process must be this. When an external object is 'perceived' the impressions upon one or more of the person's sense-organs, and through them upon related sensory centres in the cerebral cortex, suffice not only to rouse these into simultaneous conjoint activity, but also such other sensory centres as may have been previously called into action when an object of the same kind had been presented. Such a process could only be brought about by the previous growth of structural communications, of some kind, between the related groups of cells in the several sensory centres of the cortex. Where this has occurred, we may understand, in a measure, how the excitation of the cells of any one group may suffice to revive more or less strongly in other groups just such molecular changes as like objects had on previous occasions excited. Processes of this kind take place with such rapidity, that the several excitations are practically simultaneous; thus the combined effects reveal themselves in Consciousness as a single Perception of this or that external object ('The Brain as an Organ of Mind,' p. 178).

Perception in relation to ordinary processes of Thought. Herbert Spencer ('Principles of Psychology,' Vol., I. p. 187) says:—"Mental actions, ordinarily so called, are nearly all carried on in terms of those tactual, auditory and visual feelings, which exhibit cohesion and consequent ability to integrate in so conspicuous a manner. Our intellectual operations are indeed mostly confined to the auditory feelings (as integrated into words), and the visual feelings (as integrated into impressions and ideas of objects, their relations and their motions)."

Words are the symbols with which our thoughts are inextricably woven. The revived feelings or ideas of Words, may enter into thought-processes by a revival of activity in different sensory regions of the cerebral cortex. There may be a revival of sounds of words (as we hear them in ordinary Speech); there may be a revival of visual impressions of words (as we have seen them in written or printed characters); and, lastly, there may be a revival of feelings of the muscular contractions concerned in the articulation of words (as experienced during our pronunciation of such words).

Of these modes of 'ideal' recall of words, the two former are distinct and easily recoverable; while the latter is characteristically vague and difficult of conscious realisation. Let any one contrast his idea of the sound of the word 'London,' or his idea of the appearance of the word when printed or written, with his idea of the muscular and other feelings associated with the articulation of the same word, and the inferiority in definiteness and recoverability of the latter will at once become obvious.

Now it is a matter of extreme importance—for the due understanding of the several kinds of speech defect, and for the success of our endeavours to refer them to defective activity in this or that physiological region of the hemispheres—that we should definitely know in what sensory region of the cortex words are recalled to mind during ordinary thought-processes. Unfortunately, unanimity on this subject does not exist. Two distinct views, radically opposed to one another, have been advocated.

There is (1) the view that words are, in the main, revived in ordinary thought as auditory feelings; and there is (2) the view that words are revived as 'motor processes'—that is, as faint excitations of the processes occurring in motor centres during the articulation of words.

The former view is that which the writer has now for many years (since 1869) advocated. As may be seen from the quotation from H. Spencer cited above, it is the view to which he has given the weight of his authority; and that of other psychologists might easily have been added. The latter view, is the doctrine of Wundt and Bain, which, for years past, has been earnestly promulgated by Hughlings Jackson, and has lately received the strong support of Stricker. It applies to speech movements, the doctrine which they hold in regard to the memory of movements in general.

I object to the latter view on two grounds; first, because it maintains that that particular mental representative of a word, which is least distinct and most difficult to revive, constitutes, in the language of Bain, the "material of our recollection" in the use of words; and, secondly, because even if it were true that during thought words were revived not as auditory but as articulatory feelings, I should wholly reject their doctrine as to the seat and manner in which articulatory feelings are revived—to wit, that they are faint revivals of processes occurring in motor centres. The extent or fundamental nature of my opposition to this latter view may be gathered from the following quotation ('The Brain as an Organ of Mind,' p. 599):—"Motor centres, wherever they may be situated, are parts whose activity appears to be wholly free from subjective concomitants. No 'ideal' reproductions seem ever to take place in such centres; they are roused into activity by outgoing currents, and, so far as we have any evidence, the induction in them of molecular movements which, immediately afterwards, issue through cranial and spinal motor nerves to muscles, are simply physical phenomena. These [motor nervous] processes are, apparently, as free from subjective accompaniments as are the actual molecular processes which they incite in the muscle itself. It is the altered condition of the muscles thus induced, and of contiguous parts as occasioned by the movement, which together engender a body of ingoing impressions the terminus for which is the Kinæsthetic Centre." This therefore is a true sensory centre, and in it 'ideal movements' may be revived, either alone or conjointly with related Visual or Auditory Impressions.

This whole subject is so important in regard to the interpretation of Speech Defects, and also in regard to the even more important and wider question, as to whether true motor centres exist in the Cerebral Cortex, that it seems desirable to interpolate here a brief discussion of the views which I hold on these questions. It seems all the more needful to do this seeing that so high an authority as Dr. Hughlings Jackson claims that his view (of the revival of words as 'motor processes') gives "the best anatomico-physiological explanation of the phenomena of Aphasia when all varieties of this affection are taken into consideration" ('Med. Press and Circular,' Oct. 21, 1874, p. 347). From this notion I must altogether dissent.

The **SENSE OF MOVEMENT (Kinæsthesis*)**.—Just as we have a visual, an auditory, and a tactile sense, so we have a kinæsthetic sense, concerned like the others with impressions of a definite kind, viz., with those which are evoked by muscular movements.

A careful examination of the different views expressed in regard to the so-called 'Muscular Sense' (in Appendix to 'The Brain as an Organ of Mind') led to the view that this term ought to be abolished, as being in several respects misleading, when applied (as it often is) with totally distinct significations, partly referring to some and partly to all the impressions which we derive from our moving members, or from Movements generally. We may much more reasonably and conveniently, in face of all the disagreements concerning the 'muscular sense,' speak of a Sense of Movement, as a separate endowment, of a complex kind, whereby we are made acquainted with the position and movements of our limbs, whereby we judge of 'weight' and 'resistance,' and by means of which the Brain also derives much unconscious guidance in the performance of movements generally, but especially in those of an automatic type.

Impressions of various kinds combine for the perfection of this 'sense of movement,' and in part its cerebral seat or terminus coincides with that of the sense of Touch. There are included under this sensory endowment, as its several components, (*a*) a set of Conscious Impressions of various degrees of definiteness, viz., cutaneous impressions, impressions from muscles, and other deep textures of the limbs (such as fasciæ, tendons, and articular surfaces); and, in addition, there seems to be a highly important set (*b*) of 'unfelt' Impressions, which guide the motor activity of the Brain, by the information (unconscious) which they afford as to the different degrees of contraction of all the Muscles concerned in the production of any given movement.

The occurrence of Movement is for the Kinæsthetic Sense, what the presentation of an object is to the Visual Sense; and the inability to cognise the impressions occasioned by Movement (either those that are conscious, those that are unconscious, or both) which is sometimes produced by certain morbid conditions of the spinal cord or of the brain, is a defect of the Kinæsthetic Sense altogether analogous to amblyopia or blindness in relation to the Visual Sense.

The impressions coming from every one of the 'special' Sense Organs are, in part, dependent for their various combinations upon the movements of such organs, and for this, as well as for other reasons subsequently to be referred to, the connections existing between the several 'perceptive centres' for such impressions (especially those of Touch and Sight), and the Kinæsthetic Centre must be peculiarly intimate and complex.

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* From *κινέω*, to move, and *αἴσθησις*, sensation. To speak of a 'Kinæsthetic Centre' will certainly be found more convenient than to speak of a 'Sense of Movement Centre' (*see* 'The Brain as an Organ of Mind,' p. 543).

Each 'special' Perceptive Centre and also the 'visceral' Centre may, at times, and according to the nature of the stimulus, form the starting point in 'sensori-motor' or in 'ideo-motor' Acts. From one or other of these centres outgoing stimuli issue to rouse the Motor Centres. But whether these impulses pass off from such 'special' or 'visceral' Centres directly, or whether (without our consciousness) they invariably pass from them to, and then off from, some parts of the Kinæsthetic Centre must be considered to remain, for the present, uncertain—though it seems much more probable that the latter is the process which actually occurs.

On other occasions, either of the 'special' Perceptive Centres may receive stimuli which form the initial starting points of currents ending in Voluntary Acts; though the immediate execution of the movement thus prompted may, in the case of the majority of limb-movements, be dependent upon the secondarily excited guidance of co-active Visual and Kinæsthetic Centres—just as in the case of the complex movements concerned in Articulate Speech, the immediate execution of such movements is dependent upon the regulative activity of the combined Auditory and Kinæsthetic Centres (*loc. cit.*, p. 555, and chap. xxix). These particular movements seem to be related to the Auditory Centres, in just the same way that movements of the limbs are related to the Visual Centres.

In regard to what has been advanced in the last two paragraphs something further requires to be said. James Mill ('Analysis of the Human Mind,' 1829) held that what have been commonly termed 'Muscular Sense' impressions intervene, and come into operation as determining agents in the case of limb-movements at a stage immediately posterior to the Visual Idea or Conception of the movement to be executed (where such conception exists in consciousness), and immediately anterior to the actual occurrence of the Voluntary Movement. The probability that this view, enunciated more than fifty years ago, by a psychologist as remarkable for the keenness of his analysis as for the clearness and simplicity with which he expresses his views, gives a correct account of the phenomena, may be gathered from a consideration of the following facts.

Where the movements which it is desired to execute are complex and difficult, and we have to learn them by imitation of the movements of other persons, the sense of Sight is then doubly brought into play. It is necessary at the commencement, and during the continuance, of our efforts to copy such movements, to look alternately at our model and at our own moving members. A long time and much practice is, in fact, required before a person learning to dance, or to play upon some musical instrument, is able to execute either of these actions without the aid, from moment to moment, of guiding visual impressions. During the process of learning, therefore, the Visual Centre evidently exercises a dominating influence.

In time, however, the impressions pertaining to the 'Sense of Movement' (which are, of course, always associated with those of Sight) become, by way of their organized channels, sufficiently associated with them and with the newly organizing 'motor' mechanisms, to permit the movement (when once initiated) to be continued under the immediate guidance of Kinæsthetic Impressions only—that, is without further necessity for a conjoint direction through the sense of

Sight. As Jaccoud pointed out ('Les paraplégies et l'ataxie,' 1864, p. 601) the sensorium requires to learn, in the first instance, what conditions and positions of the moving parts are related to such and such tactile and other impressions coming from them. It is only at the termination of this apprenticeship that we are enabled to conclude directly from Kinæsthetic Impressions as to the precise conditions of such moving parts. This process of education can only proceed correctly by reason of the comparisons which we are accustomed to make from moment to moment, between the positions and movements of the limbs as revealed by Sight, and the sum-total of Kinæsthetic Impressions simultaneously received from the same parts.

The same different tracts of the Brain that are called into simultaneous or immediately successive activity for the initiation of any set of Voluntary Movements would probably remain in activity during the continuance of such movement, though not exactly in the same relative proportions. Thus, if we suppose the centres specially called into activity as guiding centres to be the Visual and Kinæsthetic, it may well be that the former has a dominating influence in the production of the initial Conception of the Movement about to be executed. And yet the distinctness of the Idea or Conception of the Movement (which must be partly visual and partly kinæsthetic in its origin) will be found to vary with the degree of familiarity, and consequently with the ease of execution of the Movement. In the case of the simplest voluntary movements, and those which have been often repeated, an Idea or Conception of the Movement needed, scarcely obtrudes itself at all as a conscious element of the Volition. This is a part of the process which has here become more or less latent, and which in ideo-motor and sensori-motor actions has become wholly latent; though it probably still remains even in them as one of the necessary links in the chain of causation. On the other hand, during the continuance of voluntary as well as of all varieties of automatic movements, it seems clear that the Kinæsthetic Centres exercise the supreme guiding influence. Its impressions alone—even when they very imperfectly, or not at all, rouse our consciousness as to their existence—suffice to inform us (that is, suffice to excite the proper cerebral 'centres' in ways definitely related to different positions and muscular tensions) as to the exact position of our limbs, and as to the nature and degree of their Movements.

The mode of acquisition above indicated, seems well to accord with our other interests and with the daily necessities of our lives. The sense of Sight greatly facilitates the process of learning, and its vivid impressions speedily enable the Brain to appreciate aright the more vague and occult impressions coming to it simultaneously through the Kinæsthetic Centres. Soon however the Visual Sense, which we need for so many other important purposes, no longer requires to be concentrated wholly on the performance of movements. Later still, our attention or Consciousness becomes further freed from disturbing details connected with movements. The possibly conscious impressions pertaining to the sense of Movement' at last habitually pass unheeded, and then we come to be able to perform multitudes of daily actions under the guidance of mere 'unconscious' Kinæsthetic Impressions.

Thus the working of the 'motor' side of our complex nervous mechanism, even when it is concerned in executing the behests of Will, proceeds so smoothly, and is practically so much unheeded as to leave us free to follow up the threads of our Conscious Life unhindered by the multitudinous details pertaining to the varying states of innumerable Muscles acting in ever changing combinations. The majority of us may thus be truly thankful that we have not, in reality, any such 'muscular sense' as some psychologists imagine for themselves; and rejoice that, even in Voluntary Movements, the Mind knows nothing concerning the Nerves or the Muscles by the intervention of which the processes are accomplished.

From what has previously been said it must be clear that the performance of a Voluntary Act is preceded by an Idea or Conception of the Movement which we desire to execute. This Idea or Conception is, for ordinary movements, compounded of two kinds of past Impressions, namely, those of the Visual Sense and those of the Kinæsthetic Sense. Again, it must be remembered that the Kinæsthetic Sense includes two different sets of impressions, the one set (*a*) being conscious impressions derived from our moving members (proceeding from skin, muscles, tendons and joints); the other being (*b*) unfelt impressions emanating from the muscles in relation with their varying states of contraction, and therefore affording information to the brain of the most important kind. The evidence demonstrating the existence of this last set of unfelt impressions emanating from muscles, and pointing to their importance for the guidance of muscular movements, is derived from certain carefully observed cases of diseases, as the writer first pointed out in 1869 ('On the Muscular Sense,' *loc. cit.*).

Now, in the chapter on 'The Will,' in his work already referred to, it appears that James Mill, in 1829, clearly appreciated the fact that the 'Idea of the action' to be performed is two-fold. He says, "There are two Ideas very different from one another, to both of which we give the name 'Idea of the action.'" Of these, he adds, "one is the outward appearance of the action, and is always a very obvious Idea." The other is a copy of certain internal sensations, which a few pages before he had spoken of generally as sensations accompanying the movement, and which he also more specifically defined (p. 275) when speaking of the terminal events of a movement as:—"the contraction of the muscles, with the various sensations which the action upon those organs, and the action excited in them, imply." Of these internal sensations he says, "from the habit of not attending to them, we have lost the power of attending." And then he adds,—"This last [namely the revival of such internal sensations] is by no means an obvious Idea. And the mind passes from it so quickly, intent upon the action which is its result, that it is almost always swallowed up in the mass of association. It constitutes, in fact, one of the most remarkable instances of that class of links in a chain, which, how important soever to the existence of the chain, are passed over so rapidly, that the existence of them is hardly ever recognised. . . . *This last Idea alone, is that upon which the contraction is consequent.*"

This view is then essentially that which I myself adopt, viz., that Kinæsthetic Impressions, and especially those of which we are least conscious, are the last to be revived in the cerebral cortex, anterior to, and as actual last links in, the chain of cerebral processes, concerned with and previous to the excitation of the motor centres themselves.

And now we have to approach another all-important question, which is this,—In what region of the cerebral cortex are we to look for the so-called Kinæsthetic Centres? In reply to this query I would remind the reader that I have previously expressed ('The Brain as an Organ of Mind,' 1880, pp. 522-532) my strong dissent from the notion that the several sensory centres have any such restricted localisation as that which has been assigned to them by Ferrier. I would also call attention to the fact that we are concerned here specially with part only of the impressions pertaining to the Kinæsthetic Sense, viz., with those (*b*) unconscious impressions from muscles which seem to be so intimately connected with the production of movements. It matters little, in reference to this question, what is the cerebral terminus for the other impressions pertaining to the Sense of Movement, viz., those from the skin, those from the ordinary sensory nerves of muscles, and those from the tendons and joints. It is possible that the impressions of this group may be distributed to the same parts of the cerebral cortex as those of Touch and Common Sensibility; and the hippocampal region may be, as Ferrier contends, an all-important part of this area. Admitting this, however, it does not in the least follow that (*b*) the unfelt impressions emanating from contracting muscles should proceed to the same cerebral terminus. On the contrary, we know from pathological observations made on man that these two classes of impressions, viz., those unconscious impressions from muscles and those conscious impressions from the skin and deeper textures of the limbs, pursue paths distinct from one another in some parts of their course. Their paths seem, for instance, to be topographically distinct from one another in the spinal cord; they are probably more or less contiguous in the cerebral peduncles; whilst they may subsequently diverge again and go to different, though functionally related, cerebral convolutions.

It is well to point out in regard to this difficult question, that what little can be gleaned concerning it must be obtained from the study of diseased conditions in man—from nature's experiments, that is,—since experiments upon animals can teach nothing that is distinctive in regard to this particular endowment. As I have elsewhere ('Brain as an Organ of Mind,' p. 69) pointed out, these muscular impressions "differ altogether from others [*i.e.*, from other sensory impressions], whether of external or of internal origin, by the fact, that they follow or accompany movements whose intensity they are supposed to measure, and do not of themselves incite movements." They are thus altogether different from tactual, visual, or auditory impressions, the occurrence of which is apt to lead to movements of some kind. The existence or absence of any such responsive movements in lower animals has, indeed, to be considered, when we seek to determine whether, in an animal under observation, its sense of touch, vision, or audition still persists. An altogether finer analysis is needed for detecting the loss of kinæsthetic impressions, especially those (*b*) which do not rouse our consciousness. Any such analysis is impossible without the aid of speech in the individual that is the subject of our experimentation; and even then we may be able to obtain no certain information concerning such loss, if the cortical centres for such impressions should themselves be damaged—seeing that the loss of the cortical termini for this endowment would, in accordance with our theory, cause actual motor paralysis. But, concerning this latter view we are now about to speak.

Looking to what has been said above, and supposing that these unconscious kinæsthetic impressions have that kind of immediately determining relation to the production of voluntary movements which was originally sketched by James Mill, and which I have endeavoured to enforce in the foregoing pages, let us inquire for a moment what would be the effect of irritating, and also the probable effect of the destruction of such kinæsthetic centres in the cortex of one hemisphere. Clearly the effect of their irritation, or of the outgoing fibres proceeding from them, ought to be the production of such movements as their activity is accustomed to incite; on the other hand, it should be almost equally clear that the effect of the destruction of these centres or of their efferent fibres, ought to be the production of motor paralysis in related parts of the body.

It will at once be recognised that these are exactly the results obtained by Hitzig, and afterwards more precisely by Ferrier, from irritation or destruction of those convolutions of the parietal region which border upon the fissure of Rolando. There is not the least necessity therefore for supposing that these parts of the hemisphere contain 'motor centres;' they may be merely portions of 'sensory' centres of the kinæsthetic type, if we may apply such a term to the cerebral termini for impressions from muscles, which do not reveal themselves to consciousness. Such, indeed, is very nearly the view expressed by Hitzig, and also by Nothnagel; and to this opinion I have also been driven by the considerations above adduced.

The cerebral cortex is, in my view, to be regarded as a continuous aggregation of interlaced 'centres,' towards which ingoing Impressions of all kinds converge from all parts of the body, and also of other related regions in which higher, or derivative, mental processes are carried on. Here, all the various ingoing currents evoked by the organism's relations with its outside world, and by changes in its own non-neural tissues and organs, come into relation with one another in various ways, and conjointly give rise to nerve actions, which have for their subjective correlatives all the Sensations and Perceptions, all the Intellectual, and all the Emotional Processes which the individual is capable of experiencing. From these terminal and complexly related 'end-stations' for ingoing currents, and from various annexes in connection therewith, above referred to, outgoing currents issue, which rouse in definite ways the activity of the highest 'motor-centres' (the Corpora Striata and the Cerebellum), and through them evoke the properly adjusted activity of lower motor mechanisms, or combinations of such mechanisms (in pons, medulla, and spinal cord), so as to give rise to any movements that may be desired, or which are accustomed to appear in response to particular Ideas or Sensations.

The plan on which nerve centres generally are constructed, of whatsoever grade, makes it essential that the stimulus which awakens the activity of a motor centre or ganglion, shall come to it through connecting fibres from a sensory ganglion or centre—that is, from cells which stand in immediate relation with ingoing fibres. If for instance we look at the simple nervous systems of such an animal as a Gasteropod, we may find in some of them two supra-œsophageal Sensory Ganglia connected by distinct commissures or internuncial fibres with two conjoined lower Motor Ganglia. It can scarcely be doubted that stimuli (as sequences of the nervous processes concerned with Sensations) are accustomed to pass from these Sensory Ganglia along the commissural fibres uniting them with the Motor Ganglia, and that in accordance with their different origins or starting points, these stimuli may cause the latter ganglia to evoke distinctive muscular contractions in various parts of the body. Could we galvanize separately the several sensory ends of 'internuncial' fibres we should doubtless evoke similar movements. But would such facts entitle us to infer that these supra-œsophageal Sensory Ganglia contain 'motor' centres? Assuredly not: no more than we should be entitled to call the 'sensory cells' on the ingoing side of any simple nervous mechanism for Reflex Action 'motor cells,' simply because a stimulus issues from them which ultimately evokes the Movement—that is, after it has passed through other nerve elements which, by common consent, are regarded as 'motor cells.'

The discussion just terminated may be supposed to stand as a long parenthesis. After such an aside, we may now return with advantage to our original position in the argument, and may briefly state why we wholly reject the notion that words are revived during silent thought in motor centres, as faint articulatory processes.

In the first place I would repeat that, in my opinion, not a single unquestionable fact can be adduced tending to prove that the actual processes taking place in motor centres have any subjective side whatsoever; or that they lead to anything in nerve tissues except to a downrush of molecular disturbance in a peripheral direction. True, this downrush causes contractions of muscles, and these contractions are associated with, or immediately followed by, a number of ingoing impressions (kinæsthetic). All the facts, or supposed facts, that were relied upon by Wundt or by Bain as evidence of the existence of motor feelings as concomitants of the action of motor centres or of the passage of outgoing currents have, in my opinion, and also, I may add, in that of Ferrier, proved worthless for establishing any such position; whilst, on the other hand, the notion that the feelings resulting from or connected with movements, are all of them the results of ingoing impressions from the moving parts themselves, conveyed to the brain by afferent nerves of different kinds, has now been clearly shown. This whole question has been fully discussed in an Appendix (entitled 'Views concerning the Existence and Nature of a Muscular Sense') to my work 'The Brain as an Organ of Mind.' To that Appendix I must refer the reader, for the evidence in support of the positive opinion above expressed.

This is the answer I would offer to the position assumed by Hughlings Jackson in regard to the mechanism of thought and speech processes in particular, and also in regard to his general doctrine that "mental operations in the last analysis must be merely the subjective side of sensory and motor substrata." This last dictum is one which would be perfectly legitimate, had the above-mentioned doctrine of Wundt and Bain been correct; but the two views are so closely connected that they must stand or fall together. If our cognizance of movements comes only by means of ingoing impressions and sensory processes, then movements are represented in the cerebral cortex only by sensory centres, and Dr. Hughlings Jackson's position becomes an untenable one—words are not revived in silent thought as motor processes, nor is there any reason at all for postulating the existence of motor centres in the cerebral cortex.

But, even if we adopt the more legitimate view that movements in general, as well as articulatory movements, are represented in the cerebral cortex only by sensory centres, there are still good reasons for rejecting the notion that, the 'material of our recollection,' in the use of words during silent thought, are revived articulatory sensations. This is nobody's view in particular; but it may be as well to state some of the objections that may be alleged against it, more especially as such objections would apply equally well to that other form of the doctrine which is supported by Dr. Hughlings Jackson—viz., that words are revived, in silent thought, as faint articulatory processes taking place in motor centres.

In the first place, it must be evident from what has been said in regard to the mode in which Speech is acquired by the child, that during the few months in which words enter into its rudimentary trains of thought before it has acquired the power of articulating them for itself, they must be revived as auditory impressions.

Secondly, there is, as we have seen, a much greater definiteness of impression and readiness of recall for auditory impressions than for articulatory feelings; and so far, therefore, there is a greater fitness in the former for serving as the material of our recollection of words in ordinary thought-processes.

Thirdly, there are excellent reasons for believing that revived auditory impressions continue, after the acquirement of speech by the child, to have the same relation to its thought-processes as they must have had before its acquirement of the power of speaking. If this were not so, it would be impossible to understand why total deafness supervening in a child in full possession of speech, as late as the fourth, fifth or even the sixth year, will certainly entail dumbness, or complete loss of speech, unless the child be drilled in lip-reading, that is, unless the primary incitation to acts of speech be gradually transferred from the auditory to the visual centres. On the other hypothesis, there is no reason why the supervention of deafness in a young child should, if nothing be done, certainly entail complete loss of speech.

And, lastly, the notion that words come up in thought-processes as revived articulatory feeling would be to confer upon this class of sensations a degree of importance in relation to thought-processes, which has been postulated by no philosophers or physiologists except Wundt, Bain, and a few others; and by them almost solely as a consequence of their view that such feelings are 'concomitants of outgoing currents.' But if that general view has been proved to be erroneous, then, with it must go these notions derived therefrom, as to the great importance of the articulatory feelings in thought-processes.

But, whilst altogether declining to admit the importance of revived articulatory feelings in the prosecution of silent thought, we claim for them (as sensory, not as motor processes) an all-important function when thought has to be expressed in articulate Speech.

The nature of the various Speech Defects which we shall have to describe, will be found to be capable of receiving a ready explanation in accordance with this view.

A few more words concerning the Perceptive Centres, their relations to Words, and the modes in which they are habitually called into activity, will suffice to bring our long preliminary discussions to a conclusion.

As the act of Perception involves the automatic comparison of present impressions with revived past impressions of the same kind, as well as of some or all other kinds of impressions capable of being yielded by the object perceived, it happens that, even in the simplest so-called 'Sensation,' the conjoint activity is necessitated of no one limited tract of convolutional grey matter—but rather of widely extended cell and fibre mechanisms corresponding, it may be, with many more or less diffused and complexly related Perceptive Centres (*loc. cit.*, p. 522). Seeing that each Perceptive Centre forms the basis or starting point of different processes of Ideation, and, therefore, of Thought, and that the several centres must have the same kind of relation to Emotion, we may find therein reason for a belief in the probability that the different Perceptive Centres are diffuse in seat, and that widely separated parts of the Cerebral Hemispheres are probably knitted together for simultaneous action even in the simplest sensory Perception—containing as this process does the germs of Thought and Emotion, to say nothing of 'Volition.'

Although these diffuse, but functionally unified, nervous networks may differ altogether from the ordinary conception of a 'centre' (owing to their assumed lack of topographical distinctness and exclusiveness), it is still convenient, for the sake of brevity, to be able to refer to such networks as 'Centres.'

The great importance of the due activity of the Auditory and Visual Perceptive Centres, and the absolute dependence of the great bulk of our intellectual perceptions, of our memory of words and of our powers of thought, as well as of intellectual expression, upon the functional integrity and proper inter-action of these parts, may have been gathered by the reader as strong probabilities from what has already been said.

Looking to the extremely important part which Words, either spoken or printed, play in our intellectual life, and to the manner in which they are interwoven with all our thought-processes, it becomes highly probable that most important sections of these Auditory and Visual Perceptive Centres are devoted to the reception, and consequently to the revival in thought, of impressions of Words: and for convenience of reference it is permissible to speak of these sections as Auditory and Visual Word-Centres, respectively. Similarly, there must be what we may call Kinæsthetic Word-Centres, of two kinds—the one in relation with Speech-movements and the other in relation with Writing-movements—holding a like all-important relation to the expression or communication of our thoughts, either by Speech or Writing. It is possible that the parts of the Auditory and Visual Centres in relation with Word-impressions, may be as definitely defined as are the analogous parts of the Kinæsthetic Centre in relation with Speech-movements. Certain it is, that there are some varieties of Amnesia in which the part of the Visual Centre in relation with Words seems to be specially at fault (causing 'Word-blindness'); just as there are other cases in which the part of the Auditory Centre in relation with Words is either wholly or partially inactive (causing 'Word-deafness')—in each case without apparent defect in other parts of the centre in question.

It is important to recollect that the Perceptive Centres may be called into activity in three modes, in states of health; but that departures from this rule occur in various morbid conditions, leading to this or that kind of Speech Defect. The three modes are these:—(1) By means of sensory Impressions, of external or of internal origin; (2) by 'Association'—that is by impulses communicated from another Centre, during some act of Perception or Thought-process; and (3) by 'Voluntary' recall of past impressions as in an act of Recollection. It is convenient for practical purposes to separate the second and third mode of the calling into activity of perceptive centres; still, they are probably closely related to one another, although we have no definite knowledge of the processes actually involved in the latter.

The excitability of the several Centres—that is, the molecular mobility of their constituent tissue elements, may vary much with age, state of health, or the existence of different morbid conditions. Their excitability may be so much lowered, that they are only capable of responding to powerful stimuli; so that whilst Volitional recall or Recollection may be impossible within their province, such centres may still be capable of acting in 'association' with other centres (that is in an automatic manner during an ordinary process of thought), or, at least, under the sensory stimulus or external impression which is the fore-runner and awakener of a perceptive process. At other times the excitability of Perceptive Centres may be unduly exalted, so as to lead to hallucinations, illusions, and a wholly different class of defects often met with among Insane persons.

One other point should be referred to before bringing these remarks to a close, and that is the decided preponderance of nerve processes in relation with Tactual and Kinæsthetic Impressions in the left as compared with the right cerebral hemisphere. Owing to the great preponderance of movements of the right arm and hand as compared with those of the left side, the Kinæsthetic Centres of the left cerebral hemisphere must be much better developed in the great majority of persons, than those of the right hemisphere. The impressions of the Kinæsthetic Sense are, in this respect, precisely like those of Touch—and these two kinds of sensory endowment, as we have seen, merge into one another so imperceptibly as to make it, in part, impossible to separate their cerebral centres from one another.

This preponderating activity of the left cerebral hemisphere in regard to Tactile and Kinæsthetic Impressions (about which there is no room for doubt) may have something to do with another fact to which we shall soon have to refer more in detail, viz., that the left hemisphere is the most potent and seems to take the lead in some thought-processes, and in giving rise to the voluntary excitations which determine the muscular acts involved in Articulate Speech.

If ordinary care be exercised, the three conditions, or rather groups of Speech defects, known as Aphemia, Aphasia, and Amnesia, need never be confounded with one another, at least when they exist separately.

In Aphemia, the patient knows quite well what he wants to say; there is no tendency to use words that he does not wish to employ; nor, consequently, to say what he does not mean to say: he simply has a difficulty more or less marked in the articulation of words. If he is not paralysed on the right side, his power of expressing his thoughts by writing remains as perfect as it ever was.

In Aphasia the patient commonly articulates some one or two sounds or actual words quite distinctly, but his vocabulary is limited to these. There is no indistinct utterance; rather an inability to attempt to utter any proposition, and the indiscriminate use of some one or two words, or mere sounds, on all occasions. The aphasic patient cannot repeat a single word after you—he generally cannot repeat when bidden to do so, even one of the words which he is frequently making use of. He understands everything that is said to him. His power of expressing his thoughts by writing is commonly greatly impaired—even in cases where paralysis of the right hand does not exist, or when, this disability existing, efforts have been made to teach him to write with his left hand. It is not that he writes wrong words, but that he cannot write at all, or, perhaps, can only succeed in copying a few letters.

In Amnesia there may be no difficulties in articulation; but there is often hesitation and difficulty in the finding of right words, or the substitution of altogether wrong words, or words wrongly pronounced. The amnesic patient's defects in writing are of the same order. He is often not paralysed in the right hand; he commonly writes slowly with it, using wrong words and making many mistakes in spelling. Lastly, one of the most important characteristics distinguishing Amnesia from Aphasia, lies in the fact that the amnesic patient can almost invariably repeat any word after you. Exceptions to this latter rule are only very rarely met with; so that the inability of a patient, who does utter some articulate sounds or words, to repeat a word when bidden, becomes one of the best practical indications that such a patient is suffering from Aphasia, and that this name may be correctly applied to the defect in question.

For the diagnosis of the compound conditions often met with, and referred to on p. 102, in which some two of these defects coexist, we must, of course, depend upon the recognition, in the same individual, of their particular characteristics.

Again, it should be understood that where the diagnosis of Amnesia has been made, we must by no means rest satisfied with such a diagnosis as final. Amnesia is the name of a generic state, under which are included many species. Concerning the best mode of arriving at a more specific diagnosis I shall shortly have something to say; though it will be better first to deal with the question of the localising value of the three primary conditions above described.

Aphemia from the point of view of Localisation.—The localising value of Aphemia is not great. Not much need be added to what has already been said upon this subject (p. 100). Lesions in the Medulla and in the Pons are frequently associated with the most severe forms of this defect; or there may be slighter manifestations of it with disease in the same locality. Hemiplegia from lesions higher up (that is nearer the cerebral cortex), and on each side of the brain may also be associated with minor forms of Aphemia.

In the most extreme form of Aphemia the patient may be absolutely speechless, though able to communicate his thoughts as freely as ever by writing. I am disposed to think that the most typical cases of this kind, are due to limited lesions involving the efferent fibres from the third left frontal convolution just beneath the cortex, that is the emissive fibres from the Kinæsthetic Centre for Speech-movements.

Aphasia from the point of view of Localisation.—The localising value of uncomplicated Aphasia is commonly supposed to be very great. In the first place this is a condition almost always associated with disease or injury of the left hemisphere of the Cerebrum; the exceptions being comparatively few, and in the cases in which they do exist the patients are almost invariably found to be left-handed. But we can go further, and, through the labours of Broca in the main, we are able to affirm that the existence of typical Aphasia is strongly indicative of the presence of a lesion (whatever other parts may be involved) occupying the third left frontal convolution—often, therefore, spoken of as 'Broca's convolution.'

The possibility of the occasional production of real Aphasia by lesions of the brain in parts other than the third frontal convolution, will be referred to presently under the next section (p. 122).

The simultaneous affection of the third frontal convolution on each side of the brain, produces a condition of absolute speechlessness, or complete Aphasia.

Amnesia from the point of view of Localisation.—Here we have a condition whose localising value is comparatively indefinite; it is decidedly greater than that of Aphemia, but decidedly less than that of Aphasia. It must be remembered, too, that Amnesia is a much broader or more variable kind of defect than Aphasia; and, therefore, it is not unnatural to find that defects belonging to this category should be produced by lesions extending over a wider area.

It may be said, in the first place, that Amnesic defects are always due to disease in the cortex cerebri. They are, however, not so distinctly limited to disease in the cortex of the left hemisphere as is the Aphasic condition; although it may certainly be said that, well marked Amnesic defects occur much more frequently with lesions in the cortex of the left than with those in the cortex of the right hemisphere. Still, on this subject, and as to the extent to which it holds good for different kinds of Amnesic defect, much further information is required.

If we turn now to the more specific question, as to what are the parts of the cortex in which the occurrence of disease or injury is prone to produce Amnesia, I think that a tolerably definite answer is possible. As pointed out in 1880 ('The Brain as an Organ of Mind,' p. 682), I believe that—"Lesions about the posterior extremity of the Sylvian Fissure of the left Hemisphere will probably prove almost as instrumental in producing one or other variety of Amnesia, as lesions of or about the third left frontal are of inducing Aphasia." These convolutions include the angular gyrus, the supra-marginal lobule, and the posterior half of the upper temporal convolution. It is well known that Ferrier's experiments have led him to believe that an intimate relation exists between the angular gyrus with part of the supra-marginal lobule, and the sense of Sight; and also between the part of the upper temporal convolution named, and the sense of Hearing. Though not prepared to believe that these limited regions of the cortex ought to be regarded as centres for Sight and Hearing respectively, I am quite prepared to admit that they constitute very important parts of these centres.

But, quite independently of these experiments, there have been facts of an anatomical order, known long before, pointing to the importance of the region I have indicated, as well as clinico-pathological observations, tending to show that where we have to do with marked Amnesic defects of speech, we may, with some confidence, look for a lesion in one or other of the convolutions around the posterior extremity of the fissure of Sylvius. It is well known, for instance, that the posterior third of the hinder segment of the internal capsule conveys all the sensory impressions both general and special from one half of the body, and that the fibres composing this posterior third of the hinder segment of the internal capsule (which lies between the posterior extremity of the lenticular nucleus and the posterior half of the thalamus) begin to be distributed to the convolutions in parts contiguous to the posterior extremity of the Sylvian fissure.

Whilst it seems highly probable that lesions in the situations above-mentioned, are more likely to be productive of marked amnesic symptoms when they occur in the left than in the right hemisphere; still, both facts and theory tend to support the idea that, in many cases, somewhat similar defects would be induced by lesions in corresponding parts of the right hemisphere.

It will be found that many such cases are already on record—one of the most typical being that of Marcou as given by Trousseau. And, if we bear in mind that corresponding sensory centres in the two hemispheres are almost habitually called into simultaneous activity, and are probably in structural continuity with one another through the corpus callosum, it might be expected that irritative or destructive lesions of the Auditory or of the Visual Word Centres of the right side could scarcely occur without producing distinct derangements, at all events for a time, in the functional activity of the similar centres in the left hemisphere—which, as one is bound to admit, seems to take the lead in the expression of thought by speech or writing. It might fairly be expected, perhaps, that Amnesia produced by a lesion of the right side (in a person who is not left-handed), would be more temporary than if it had been occasioned by a similar defect in the left hemisphere.

We must now call attention to another point in connection with Amnesia and Aphasia which is of great importance from the point of view of regional diagnosis.

The condition of Amnesia may merge by insensible gradations into one of Aphasia—so that the latter state with certain extra peculiarities, may at times result from a lesion situated altogether away from the third left frontal gyrus, if, as we at present suppose, the region in which lesions have the greatest tendency to produce one or other of the forms of Amnesia is that lying around the posterior extremity of the left Sylvian fissure.

This may be easily understood. Suppose a person to be suffering from a defective activity of the Auditory Word Centre, so that Names cannot be recalled 'voluntarily' or by 'association.' There would from this cause be great hesitation and difficulties in the expression of thought, both by Speech and Writing. But suppose this mere defective activity were to be replaced by actual destruction of the left Auditory Word Centre, words could then, of course, neither be recalled voluntarily nor by association; and, still further, spoken words could not be perceived, and consequently could not be imitated. An individual thus affected would neither be able to Speak nor to Write, that is he would be completely Aphasic—with the superadded peculiarity that he would not readily comprehend spoken and perhaps written language. The latter ability might persist to some extent, because the molecular equilibrium of the Auditory Word Centre and of the related Visual Centre of the opposite Hemisphere might not be sufficiently disturbed to prevent all apprehension of spoken or of written symbols. We might, in fact, have in such a case the production of a complex Aphasic condition almost precisely similar to that met with in a girl whose case was recorded by Bazire, or even of one recorded by the writer ('The Brain as an Organ of Mind,' pp. 653 and 655)—yet such an Aphasic condition might, and would probably, have been caused by a lesion far away from the third left frontal gyrus (Fig. 7, *a*). It might be only needful for it to destroy the hinder part of the left upper temporal convolution (Fig. 7, *c*).

From what has been said above, it would seem to follow in the plainest way that ordinary typical Aphasia ought also to result from destructive disease situated anywhere between the Auditory Word

Centre, and the Kinæsthetic Word Centre in the third frontal convolution—that is, from any lesion which destroys the commissural channels between these two centres in any part of their course (Fig. 7, *b*). This from the point of view of 'cerebral localisation' is a most important conclusion, necessitating a considerable change of doctrine in regard to the pathogenesis of Aphasia.

Supposing the most important part of the Auditory Centre to be situated near the posterior end of the fissure of Sylvius (possibly in the upper temporal convolution), then the conclusion I have arrived at is that complete Aphasia may result from a lesion there, from a lesion in any part of the commissural tract between this centre and the Kinæsthetic Speech Centre, as well as from a lesion in this latter centre itself in the third frontal convolution (Fig. 7). I would further maintain, that a lesion which destroys these commissural fibres in any part of their course should produce an Aphasia of a typical kind, in no way differing from that caused by destruction of the Kinæsthetic Centre itself in the third frontal convolution.

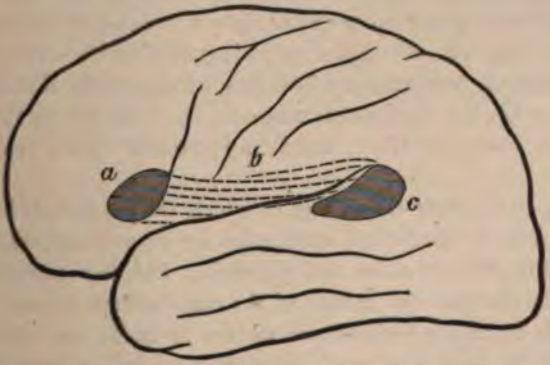

FIG. 7. DIAGRAM SHOWING THE POSSIBLE LOCATION OF AN APHASIA-PRODUCING LESION, EITHER IN THE KINÆSTHETIC CENTRE (*a*), IN THE AUDITORY CENTRE (*c*), OR SEVERING THE COMMISSURAL FIBRES BETWEEN THEM (*b*) IN ANY PART OF THEIR COURSE.

It will be for future workers to decide whether clinico-pathological evidence will support this view or not. It is one which is not at all indicated by the views of Hughlings Jackson since he supposes that (*c*) is a motor centre wherein words are primarily revived in thought in the form of faint or 'suppressed articulations.' The establishment of the extra localisations for Aphasia in situations (*b*) or (*c*) would, therefore, strongly tend to support my doctrines as opposed to those which he puts forward in explanation of the Aphasic state. The evidence at present existing in support of these extra localisations is by no means inconsiderable (see section on 'Lesions in the Rolandic Region').

Aphemia (that is, mere loss of speech) could not be produced by a lesion in this posterior Sylvian region of the brain, although pure Agraphia accompanied by 'word-blindness' might result from a lesion of the left Visual Word Centre—a combination of defects presented by a patient now under my care. The reason why mere Aphemia could not result from a lesion of the Auditory Word Centre productive of 'word-deafness' is due to the fact that a destruction of this centre would prevent the revival of words in thought which is a process that must precede spontaneous writing. Thus the double condition Aphasia (or an approximate state in which imitative writing only is possible), would necessarily result, instead of the more special Aphemic state.

From what has been said above, it will be seen that a general law may be provisionally formulated, as a working hypothesis, viz., that the tendency to mental impairment with Aphasia, and the degree of such impairment, will, other things being equal, increase as lesions of the left Hemisphere, which produce Aphasia, recede in site from the third frontal convolution and approach the occipital lobe.

Owing to the very varied way in which the several kinds of defect in the power of intellectual expression by Speech and Writing are combined in different cases, and yet owing, also, to the importance of exactly determining the nature of the combinations presented, it will be seen to be highly desirable, if we are to frame a correct regional diagnosis, to submit all such cases to a complete examination in accordance with some uniform and definite scheme. By adopting such a course we may ascertain pretty accurately the nature of the disabilities from which our patient is suffering; and, at the same time, those who are not accustomed to make such examinations may more readily assure themselves that no important points, which ought to receive their attention, have been accidentally passed over. It is found only too often that in reports of different cases of an Aphasic or of an Amnesic type, the condition of the patient in reference to one, two, or perhaps several important points, receives no mention at all; and thus for lack of information positive or negative in regard to this or that capacity, the record of the case is greatly diminished in value for all scientific purposes.

Such a scheme I now append. It is based upon the physiological views in regard to Speech Defects which have been above expressed; but it is, I believe, sufficiently comprehensive to bring out all the facts which it is important to ascertain in reference to such cases, so far as our knowledge at present takes us. It has been used for the last four or five years by my House-Physicians in University College Hospital.

SCHEMA FOR THE EXAMINATION OF APHASIC AND AMNESIC PERSONS.

Auditory Perceptive Centre, with its afferent and efferent fibres.

1. Hearing—good or bad? 2. Comprehension of Speech. 3. Appreciation of Vocal Music. " Instrumental Music.	*To test the functional activity of afferent fibres and of the Centre itself.*
4. Speech (a) Imitative. (b) Associational. (Repetition of numerals, alphabet, days of week, &c.) (c) Spontaneous.	*To test the functional activity of the Centre and of its efferent fibres.*

Visual Perceptive Centre, with its afferent and efferent fibres.

1. Sight—good or bad? 2. Comprehension of printed or written Words. 3. Recognition of numerals or Letters. (As judged by ability to point them out.) 4. Recognition of common objects. 5. Recognition of pictures of common objects.	*To test the functional activity of afferent fibres and of the Centre itself.*
6. Writing (a) Imitative. (Copying of numerals or letters, or 'transfer copying' of letters or words.) (b) Associational. (c) Spontaneous.	*To test the functional activity of the Centre and of its efferent fibres.*

Commissures between the Auditory and the Visual Centres (Visuo-auditory and Audito-visual).

(1.) Naming at sight common objects, numerals, letters, or words.
Reading aloud.

(2.) Writing from dictation, numerals, letters, words, or propositions.

After what has been already said this schema will require no explanation so far as its general purpose is concerned; some few comments as regards special points will, however, be desirable.

Where spontaneous (or 'voluntary') speech or writing is either absent or greatly impaired, the capacity which remains for exciting these acts by strong trains of association may be tested, by making the patient repeat or write the common numerals, up to ten or twenty; or the letters of the alphabet, after initial prompting; or else some form of words which he is likely to have known by heart—the Lord's prayer, for instance.

It will often be found that great differences exist between the patient's power of reading or writing figures and letters respectively; he may often be able to read or write numerals, whilst he cannot do the same with letters. This difference probably depends upon the fact that there are only nine simple numerals, whilst there are twenty-six letters in the alphabet. The patient's knowledge of or ability to deal with figures may, therefore, have become more deeply graven and automatic, than it is for letters.

Where patients cannot write spontaneously and we wish to test their ability to copy, this may be done at first by writing certain letters or words and bidding the patient copy them. If he is able to do this, he may then be tested with a more difficult task. He may be shown a printed word or sentence, and asked to copy it in writing characters—a process which for the sake of brevity is spoken of as 'transfer copying.'

A little reflection will make it probable that there must be two sets of commissures between the Auditory and the Visual Word Centres; the one for transmitting stimuli from the Visual to the Auditory Centres (visuo-auditory fibres), as in the acts of reading aloud or naming at sight; the other for conveying impressions in the opposite direction, *i.e.*, from the Auditory to the Visual Centres (audito-visual fibres), as in the act of writing from dictation. There are good clinico-pathological reasons for believing that there are really two distinct sets of fibres and not one set conveying impressions now in one direction and now in another. I have recorded one case of amnesia in which both sets of fibres, judging from clinical evidence, seem to be damaged; Broadbent has published one, in which it seems probable that the visuo-auditory fibres only were damaged; whilst a former pupil, Dr. Dingley, has submitted the notes of a case to me in which it would seem that the visuo-auditory fibres were intact, whilst the audito-visual fibres were cut across or otherwise damaged ('Brain,' Jany. 1886).

It seems highly probable that in the majority of cases, the order of events in reading aloud or in writing from dictation is such as I have above indicated. Thus, in reading aloud, the stimulation of the Visual Centre rouses, by way of the visuo-auditory commissure, related portions of the Auditory Centre and from it, as a general rule, incitations pass to the Kinæsthetic Speech Centres. Some cases of speech defect are, however, scarcely explicable except upon the supposition that Visual incitations may at times pass direct to the Kinæsthetic Speech Centres during the act of reading aloud. I have elsewhere quoted and referred to cases of this kind ('Brain as an Organ of Mind,' pp. 624, 644).

Again, in writing from dictation the ordinary process seems to be what has been indicated above, namely, that the stimulation of the Auditory Centre rouses, by way of the audito-visual commissure, related portions of the Visual Centre, and that from this, incitations pass to the Kinæsthetic Centres in relation with Writing movements. In exceptional cases, however, it seems to be possible that stimuli may pass direct from the Auditory Centre to the Kinæsthetic Centre in relation with writing movements. How else are we to explain a remarkable case of Westphal's, quoted in abstract by Kussmaul? Of this patient it is said:—"He could write very well from dictation, but shortly after he was unable to read the words he had written, and he suffered in general from complete alexia [*i.e.*, inability to comprehend written symbols]. By means of a stratagem, however, as he himself very clearly explained, he succeeded in reading the word he had written from dictation upon the tablet. He passed his finger over each letter of the written word, as if he were writing it again, and read it while so doing. He then made a sort of calculation and counted off the sum of the separate letters." It is unfortunate that we are not told whether this patient could copy writing; seeing that he was unable to comprehend written characters the probability is that he could not, and the same fact would make it highly improbable that his Kinæsthetic Centre for writing could have been effectively stimulated from such a very defective Visual Word Centre; and if that were so, we should be compelled to assume that it must have been stimulated directly by the Auditory Centre. This latter supposition seems to be strengthened by what is said concerning the stratagem by which alone he could read what he had just written. During this stratagem, it would appear, that the reverse process was executed; that is, the Kinæsthetic Impressions from the Writing Movements which he repeated, probably roused related parts of the

Auditory Word Centre, and the revival of activity in them was followed by the production of Speech-movements in the ordinary way.

In regard to some of the principal distinctions met with between different cases of Amnesia, a few general tentative remarks may here be made.

In the slighter forms of Amnesia patients experience a difficulty in recalling the names of persons, places and things, or they misapply them. It is rarer to find such defects extending to substantives generally, and to other parts of speech. It is the most 'special' associations which are the first to show signs of weakness or failure. It is reasonable to suppose that the seat of all such associations would be in the Perceptive Centres.

With other parts of speech the case may be quite different. As Broadbent truly observes ('Med. Chirurg. Trans.,' 1872, p. 192), "Words other than names, such as adjectives, verbs, etc., constituting the framework of a sentence or proposition, stand on a different footing; they are not associated with and tied down by visual, tactual, and other perceptions. Their use implies a previous knowledge of words as names, and marks a step beyond the act of naming. . . . . . They are not substantive intellectual symbols, but intellectual agents, instruments and products of intellect in action, not presentations impressed upon it. It is with respect to this class of words that it may be strictly said that 'we think in words,' for we often think [in part] in revived visual impressions not reduced in words. The convolutions concerned in their employment, will be such as are the seat of the intellectual operations."

Even though we do not quite agree with Broadbent in supposing that Intellectual Action and its Centres can be so distinctly separated from Perceptive Action and its Centres; or, in regard to the divisions which he seeks to establish between these modes of activity; or, with his explanation of the process of naming—still we cannot help thinking that what he says above is very suggestive in regard to possible differences of seat in the organic substrata for Words, according as they do or do not denote external objects. It is reasonable to suppose that names of objects might be in more immediate relation with Perceptive Centres; whilst general and abstract nouns and other parts of Speech would be much more intimately associated with regions where perceptive processes become merged into more complex, abstract, and strictly Intellectual Operations.

If this distinction be a good and valid one, and if the inability to recall names, or the miscalling of persons, things, or places, are to be considered as defects going with injuries to or altered states of Perceptive Centres, we can easily understand that they may exist with comparatively slight impairment of Intellectual Activity. And this is found to be the case.

On the other hand, the more extreme forms of Amnesia, in which wholly irrelevant propositions or a mere jumble of words are uttered, are more likely to be associated with marked impairment of Intellectual Power—to be dependent, in short, upon injuries or altered states of parts of the Brain more specially concerned with such modes of activity. We know nothing from clinico-pathological evidence on this subject, at present; hence the importance of a careful and thorough examination, both during life and after death, of any well marked and subsequently fatal cases belonging to this category.

The process of Thought seems to be in a measure independent of the Words in which the thought is expressed, so that perhaps we think in words somewhat less than is generally supposed. Its partial independence appears indicated by the very fact that we 'select' our expressions. Thus, according to the different shades of meaning sought to be conveyed in our propositions, we often deliberatively weigh or 'select,' the abstract nouns, adjectives, and verbs, that we deem most expedient for the complete communication of our thoughts to others. The process by which such words are associated with our thoughts, seems to be a little less automatic than that by which external objects, or the 'ideas' of such objects, associate themselves with words.

In the 'inco-ordinate' defects of different grades (that is, where mistakes in words are made), the defective association occurs either in the Perceptive Centres alone, or in these as well as in the parts principally concerned with more abstract Intellectual Operations. In the latter cases, Speech may be reduced to a mere meaningless jabber of misplaced words; or else to a meaningless string of articulate sounds not representing Words at all. These are the most extreme forms of such defects, and they are much rarer than the many minor forms of speech defects.

In one class of cases, the person whose Speech is reduced to a mere meaningless jabber, also does not understand what is said to him; whilst in another, though only himself able to utter a senseless gibberish, the affected person may clearly understand everything that is said to him. In the former class there is a profound mental defect; in the latter there may be little or no mental defect, such patients being often able to write correctly, and thoroughly understand what they read.

Similarly extreme defects are also noted at times in the patient's power of expressing his thoughts by means of Writing. In some cases the power of writing is reduced to the production of a mere meaningless assemblage of letters, whilst there is at the same time an inability to comprehend written or printed words. In other cases this latter peculiarity may be absent.

How far defects in the action of the Kinæsthetic Word Centres in connection with Speech and Writing respectively, or some defective association between them and the corresponding Auditory or Visual Word Centres, may be the cause of some of these extremely incoordinate defects in Speech and Writing, it is difficult to say, but it may be well to bear such disordered conditions or relations in mind as possible causes.

Much more work is needed before we shall be in a position to express a positive opinion upon this and upon many other points in connection with Amnesia, but we feel assured that the careful investigation of such cases during life, so as to be able to come to an accurate diagnosis as to the exact nature of the defects existing (so far as this may be possible); together with a precise determination of the seat of lesion in fatal cases, will ultimately enable us to make real progress in a task of extreme difficulty—that is, in the endeavour to ascertain what kind of functions are principally carried on in different regions of the cerebral cortex.

VI. CONTROL OVER SPHINCTERS.

The absence of control over sphincters is a sign of comparatively small localising value. It is a condition rarely met with where disease exists only in one hemisphere, unless the disease be of such a nature as to produce great increase of intra-cranial pressure, and consequently such an interference with the functions of both hemispheres as to lead to distinct mental impairment. In cases of this type it may be met with.

Again, it is frequently present where structural disease coexists in both hemispheres, or (what amounts to almost the same thing) where disease exists in the motor tracts on each side of the middle line in affections either of the pons Varolii or of the medulla oblongata.

Thus, this sign is met with in brain disease either (*a*) where the individual is in a condition so obtuse as not to heed his natural wants, or not to feel called upon to exercise any control over them; or else (*b*) in cases in which, though he may be fully sensible, he is unable to exercise adequate cerebral control over the spinal centres connected with the bladder and rectum, because of some morbid implication of the cerebral motor tracts on both sides.

Of course what is above said has reference to cases in which all symptoms of coma and insensibility have either been absent or, if previously existent, have passed away.

The only exceptions to these rules which I have ever met with, have been two cases in which after death similar lesions were found, viz., hæmorrhage into one of the optic thalami.

VII. DIFFICULTIES IN DEGLUTITION.

As a rule, difficulties in deglutition occurring in the course of brain disease, would point distinctly to an implication either of the pons Varolii or of the medulla oblongata. The interference with the functions of these parts may be either direct or indirect; that is, due to lesions actually situated therein, or else to lesions in other parts which cause an undue pressure upon the pons or the bulb.

VIII. CEPHALALGIA AND VOMITING.

The localising value of *Cephalalgia* is very indefinite.

When occurring as a result of organic disease, marked and persistent cephalalgia is met with principally in association either with a meningitis, or with a new growth of some kind within the cranium.

In the case of meningitis it may be either a lepto-meningitis or a pachy-meningitis; in the former case, the headache that may be met with has no localising value; whilst in the latter, both the morbid process and the severe and more or less constant pain with which it is often associated are, in the majority of cases, similarly localised. The most frequent instances of this occur in the pachymeningitis associated with syphilis.

In regard to the association of cephalalgia with tumour of the brain, it may be stated first of all in general terms that headache is apt to be most marked where the new growths either actually involve or distinctly press upon some portions of the meninges. It is not necessary that the growth should itself touch the meninges in order to produce this result; it suffices for them (*e.g.*, the tentorum) to be put upon the stretch by the bulging of brain tissue caused by some subjacent growth. This occurs not unfrequently in cases of tumour of the cerebrum and of the cerebellum.

In other cases the pain associated with tumour of the brain may be rather an indirect result of a general increase of intra-cranial pressure; and then its localisation may be variable, or at all events show no constant relations with the seat of the tumour. For instance, although in many cases of tumour of the cerebellum it happens that severe occipital headache shows itself as a prominent symptom, it also happens in some cases of this disease, that pain in the occipital region has been absent, though a severe pain in the frontal region has been constantly present.

In some rare cases where internal parts of the cerebrum have been the seat of the new growth, and where the growth does not attain any very great size, it may run its course without the occurrence of any marked cephalalgia.

The *Vomiting* of cerebral disease frequently presents itself with distinctive characters. It is often sudden and abrupt, the contents of the stomach being quickly voided without much, if any, preliminary retching or nausea; it may occur at altogether irregular times, rather than at some regular period after ingestion of food; and, lastly, in cases where headache coexists, it is very apt to be associated with exacerbations of this—especially with those exacerbations which, in cases of intra-cranial new growth, are so prone to occur on awaking in the morning.

Vomiting is also a symptom which has no distinct localising value. It is apt to be associated with the presence of tumours in all parts of the intracranial cavity, though this symptom is more especially common where the new growth is situated beneath the tentorium—either in the cerebellum, the pons, or the medulla oblongata. Where it occurs in connection with various forms of meningitis, which is not unfrequently the case, no localising value attaches to it beyond what is indicated by the name of the affection.

Apart from the period of onset, when it is of common occurrence, vomiting is rarely met with in association with hæmorrhage or softening of the brain, unless these lesions exist in the pons or in the medulla oblongata.

IX. OPTIC NEURITIS AND OPTIC ATROPHY.

Optic neuritis, although a sign of the highest importance in brain disease, is one which is quite devoid of localising value. The nearest approximation to such a value is derivable from the opinion entertained by many that some forms of it are especially prone to be induced in association with a basal meningitis—the optic neuritis being supposed to be set up by a 'descending neuritis.' But even the extreme advocates of such a view are compelled to admit that optic neuritis may coexist with tumour or abscess in any part of the brain, and that these are its common primary causes. They believe, however, that such a primary cause is, at some stage of its existence, apt to be associated with more or less of basal meningitis, and that this is the proximate cause of the optic neuritis which follows.

This is not the place to enter into any discussion of the many theories which have been put forward in regard to the causation of optic neuritis. We would only say that one form of the condition, viz., that known as 'choked disc' seems to be often caused by extreme mechanical congestion of the optic disc; and that this form cannot always be discriminated during life, with the opthalmoscope, from others which may have a more distinctly inflammatory mode of origin. Tumours, abscesses and other adventitious products within the cranium, which cause more or less excess of intra-cranial pressure may entail, in one or other mode, a mechanical congestion of the optic nerves. This may occur early or late; it may be comparatively transitory or permanent.

Similarly, optic neuritis may be an early or a late sign associated with intra-cranial new growth; it may be transitory, may persist till death, or may gradually change into a condition of white atrophy.

White Atrophy of the Optic Discs is also a sign that has no localising value in brain disease. As above indicated, it is often a remainder, or rather a sequence, of a past and well-marked optic neuritis.

In other cases it is not preceded by optic neuritis, and shows itself as a primary atrophic process, springing up in one or both optic discs. Such primary atrophies of the optic discs are, perhaps, most commonly caused by processes of sclerosis, in association with similar processes in the cord or brain (locomotor ataxy or disseminated sclerosis).

Occasionally an atrophic process of this kind occurs in the optic nerves, one or both, as an isolated phenomenon, that is unassociated with similar processes elsewhere.

X. AMAUROSIS, AMBLYOPIA, HEMIOPIA, AND DIPLOPIA.

(1). *Amaurosis* is, for the most part, a sequence of optic neuritis, or a result of secondary sclerosis, or of some primary atrophic process invading the optic nerves, such as we have referred to in the last section. From what has been there said, it will be seen that this is a sign having a very low localising value.

(2). *Amblyopia* may occur in both eyes as a mere weakening of sight associated with generally impaired health. In such cases it may, therefore, be altogether independent of intra-cranial disease.

Where optic neuritis has existed long, and in such a degree as to lead on to the sequence of optic atrophy, there must, of course, be periods of comparatively weakening sight, as the degeneration progresses and before vision is completely lost. Here, again, the amblyopia is as devoid of localising value as was the precursory optic neuritis.

Unilateral amblyopia has, however, a very distinct localising value when it is associated with a hemianæsthesia of the same side of the body. This condition of things is met with in what is called cerebral hemianæsthesia, a state that will be presently described, which is due either to structural or to functional disease in the posterior third of the hinder segment of the opposite internal capsule (see p. 152). With this form of amblyopia, ophthalmoscopic examination shows no changes in the fundus of the eye; and the pupil remains sensitive to light, because the defect occurs in the visual tract beyond the corpora quadrigemina, and these contain the reflex centres upon which the activity of the pupil depends.

This crossed amblyopia occurs in different cases with varying degrees of intensity, whether it be the result of structural lesion or of a mere functional disturbance in the region indicated. It is, moreover, especially worthy of note that precisely the same kind or range of defect may occur in each set of cases; and consequently, so far as the mere amblyopia is concerned, it furnishes no ground for distinguishing between the hemianæsthesia of structural and that of functional origin.

The phenomena presented in these cases have been thus described by Charcot.

(*a*). More or less pronounced diminution, or, what is much more uncommon, even absolute loss of the visual faculty in the eye on the same side as the hemianæsthesia.

(*b*). A normal appearance when examined by the ophthalmoscope of the optic disc and the retina. A comparative examination of the fundus on both sides, reveals no appreciable difference in regard to vascularity.

(*c*). Visual acuity, when tested in the ordinary way, is frequently found to be reduced by one half or even more.

(*d*). Concentric and general contraction of the visual field, commonly exists.

(*e*). Lastly, there is also a concentric and general contraction of the visual field for colours. It has been ascertained by Landolt, however, that this contraction of the visual field for colours occurs in both eyes, though "it is incomparably more pronounced in the eye corresponding to the side affected with anæsthesia." This peculiarity was first found to hold good for the functional or 'hysterical' form of the disease; but it has since been ascertained to obtain also in cases of cerebral hemianæsthesia originating with organic lesions.

In reference to this last characteristic (*e*) some explanations, to which Charcot directs attention, are needful.

In the normal state all parts of the visual field are by no means equally endowed with colour perception. There are some colours for which the visual field is physiologically more extensive than for others, and such differences in the relative extent of the several fields are constant in different persons. Thus the visual field is always most extensive for blue; then come, in succession, yellow, orange, red, and green; lastly, violet is perceived only by the most central parts of the retina.

In cerebral hemianæsthesia these normal characteristics regulate the mode of contraction of the visual field for colours; so that the circles corresponding with the different colours become concentrically narrowed, and, therefore, lead to many variations in the visual field for colours in different patients, according to the extent of development of this crossed amblyopia. First, the circle for violet becomes contracted to nothing, so that the patient fails to perceive this colour; then, as the disease progresses, green, red and orange follow in turn; the perception of yellow and blue persisting until the last. Finally, in the most advanced stages, no colours can be detected—tinted objects then presenting to the patient an appearance in monochrome similar to that of a sepia drawing.

Previously to the publication of these views by Charcot and others, the doctrine of von Græfe had been commonly entertained, to the effect that absolutely unilateral lesions of the cerebrum do not cause crossed amblyopia, but rather lateral homonymous hemiopia. Thus, according to this theory, a circumscribed cerebral lesion on the left side would give rise to suppression or obscuration of the right half of the visual field, and inversely in the case of a lesion of the right hemisphere. Without denying that this may be the case when lesions exist in some parts of the hemisphere, it seems quite clear that it is not so when the lesion affects the posterior third of the hinder segment of the internal capsule. Charcot seems fully to have substantiated his proposition that—Lesions of the cerebral hemispheres producing hemianæsthesia likewise determine crossed amblyopia, and not lateral hemiopia.

One of the most important results attendant upon the investigation of this subject has been the discovery of the identity of the clinical symptoms met with in what is called hysterical hemianæsthesia, with those encountered when organic disease implicates the posterior third of the hinder division of the internal capsule. Such a coincidence cannot fail strongly to support the notion that in the former more or less temporary condition, we have to do with a functional defect in the brain region just indicated.

According to Raymond, again, a hemianæsthesia with crossed amblyopia of the same type has been several times met with in persons affected with lead poisoning, who have nevertheless been cured—cases, therefore, in which it may also be supposed that the malady was not due to an organic lesion.

(3). *Hemiopia.*—Of the several varieties of hemiopia, some are fairly common, whilst others are very rare. Great uncertainty and difference of opinion exists at present as to the localising value of the different kinds of hemiopia—a difference of view dependent solely upon the divergent notions existing as to the course of the fibres of the optic nerves in two parts of their course, viz., between the optic commissure and the mesocephalic ganglia, and again between these bodies and the cerebral cortex. It is absolutely essential, therefore, that something should be said concerning these anatomical questions before speaking of the several varieties of hemiopia.

It is well known that in fishes, amphibia, reptiles, and birds, the optic nerves undergo a complete decussation at the optic chiasma so that all the impressions from each eye go to the opposite optic lobe.

Some believe, even to the present day, that a similarly complete decussation of the optic nerve fibres takes place at the chiasma in man; others altogether reject this notion and adopt the view originally propounded by Newton, and subsequently reinforced by Wollaston (who thus sought to account for hemiopic defects of sight) that at the chiasma there occurs a semi-decussation only.

Neither view can be said to be definitely proved to the exclusion of the other, and it seems quite possible that these discrepancies in doctrine may be dependent upon the fact that the course of the optic fibres is not the same in all men. If reasons can be adduced in support of such a belief, it may be supposed that differences in the degree of completeness of the decussation of the optic nerves exist in different persons, just as we now know (through the researches of Flechsig) that frequent variations, from what may be considered the normal amount of decussation of motor fibres in the anterior pyramids of the medulla, are met with when any large number of brains are examined. The frequency of anomalies in the distribution of some of the principal bloodvessels in the human body, should of itself suggest the possibility that similar anomalies may occur, at times, in the distribution of bundles or tracts of nerve fibres.

But, putting aside mere suppositions, let us come to the mention of some facts. In his article 'Optic Nerves' in Todd's 'Cyclopædia of Anatomy and Physiology,' Dr. Robert Mayne says:—"In the Museum of the Richmond Surgical Hospital, Dublin, the writer has seen specimens of atrophied optic nerves in man which furnish the most contradictory evidence upon the subject under discussion. The preparations alluded to were cases in which one eye had been destroyed either by local disease or by accident, many years previous to death, and where, in consequence, the corresponding optic nerve became wasted from disuse, while the other optic nerve continued healthy. In the majority of these specimens the wasting has been propagated backwards to the opposite tractus opticus and has implicated that structure while the corresponding tractus has been spared (see Fig. 8, A); in some examples both tractus optici have suffered a diminution of size, and in general to an unequal amount; and in one very remarkable instance the tractus opticus of the same side with the shrunken nerve has dwindled into a narrow band, while the other retains fully its normal dimensions (Fig. 8, B)."

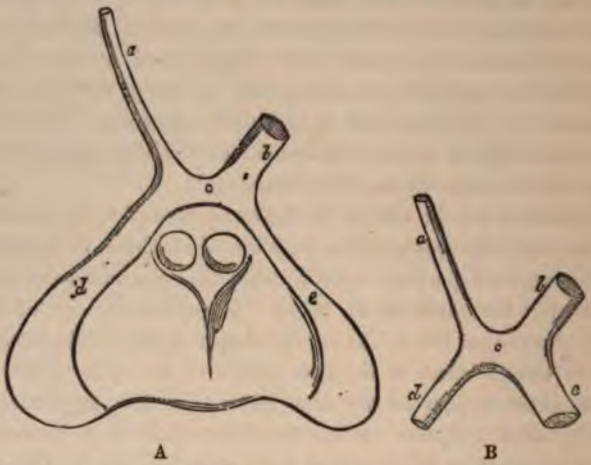

FIG. 8. ATROPHY OF ONE OPTIC NERVE, THE CONSEQUENCE OF LONG CONTINUED DISUSE. (From preparations in the Museum of the Richmond Hospital, Dublin.) [After R. Mayne].

(A)—*a*, Right optic nerve in a state of atrophy; *b*, left optic nerve healthy; *c*, chiasma; *d*, right tractus opticus healthy; *e*, left tractus opticus wasted.

(B)—*a*, Right optic nerve atrophied; *b*, left optic nerve healthy; *c*, chiasma; *d*, right tractus opticus wasted; *e*, left tractus opticus healthy.

Dr Mayne did not from these facts draw such an inference as I have indicated above in regard to the varying distribution of the fibres in different cases, he rather drew the untenable conclusion that "arguments derived from the pathology of the optic nerves can be but of little value, since they have been relied on in turn by the framers of each hypothesis as affording proof of their own peculiar views." As a matter of fact such pathological evidence is of the utmost value to those who know how to interpret it, that is, to those who trust to it rather than to some exclusive theory, adherence to which may tempt them to explain away, or to gloss over, facts not in accordance therewith.

Two pathological specimens have fallen under my own observation which also tend to show that in them the decussation of the optic nerves must have been complete or nearly so. The celebrated mathematician and logician De Morgan chanced to have been blind, almost from birth, in the right eye. At the autopsy, made in concert with Dr. Wilson Fox, I carefully examined the brain of this distinguished man. The corresponding optic nerve was atrophied, and so was the optic tract of the opposite side, whilst the optic tract of the same side presented a perfectly healthy appearance. We have likewise a specimen of a similar kind in the Museum of University College (No. 2,090), showing that in the person from whom it had been taken there had been a total and not a semi-decussation of the optic nerves.

On the other hand, some four years ago I presented to the Museum of the College another specimen (No. 5,546), taken from a patient who had died in the Hospital, which showed that in this particular individual a semi-decussation of the optic nerves had existed. The man was a worker in the Woolwich Arsenal, whose left eye was extirpated on account of an accidental injury five years before his death. At the autopsy, I found the corresponding optic nerve to be notably smaller and greyer than its fellow, but beyond the commissure the optic tracts throughout their whole extent showed no appreciable difference in size or colour. The atrophy which, doubtless, existed, was probably equally divided between them.

Those who believe that a semi-decussation is the common arrangement, favour the following hypothesis in regard to the distribution of the fibres of the optic nerves :—" Each tractus opticus sends some filaments across the chiasma to form the inner part of the opposite optic nerve, while its outer filaments continue on to form the outer part of the optic nerve of its own side; the same arrangement of the filaments prevails to the retina, so that the right side of each retina comes from the right tractus opticus, and the left side of each retina from the left tractus opticus; if then, in vision, the pictures of an object be depicted simultaneously either on the right side of the two retinæ, or on the left side of the two retinæ, the impressions in either case will be transmitted to one and the same tractus opticus."

A quotation from Charcot will, however, serve to illustrate the divergence of opinion that still exists in regard to this subject of the distribution of the optic fibres at the chiasma and in the optic tracts. After explaining the semi-decussation theory he says :—" It must not be forgotten that this arrangement of the optic nerve fibres is, anatomically considered, quite hypothetical. Although several authors, among others, Hannover, Longet, Cruveilhier, Henle, and still more recently Gudden, are persuaded that they can support it on anatomical grounds, there are others such as Besiadecki, E. Mandelstamm, and Michel, who, in opposition appeal to arguments of the same nature, and endeavour to show that the fibres of the optic nerves undergo complete decussation at the chiasma, even in man. . . . In short, we must confess that the question at the present time is far from being settled. I repeat, therefore, that the semi-decussation is to be looked upon simply as a theory which, however, explains the facts observed in clinical medicine, and is certainly superior to the hypotheses which some have endeavoured to substitute for it."

Charcot, further, believes that the optic fibres which have not decussated in the chiasma undergo, nevertheless, a decussation in the corpora quadrigemina; so that, in accordance with this view, the optic fibres which, within each hemisphere, make for the cortex have proceeded solely from the opposite eye. His views, as well as the mode of production of the different kinds of hemiopia in accordance therewith, may be understood by reference to the following diagram (Fig. 9).

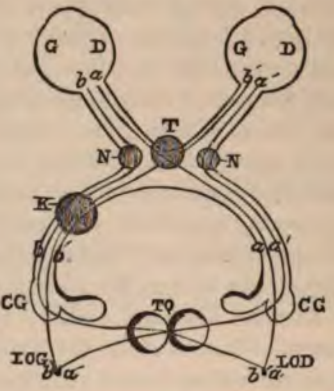

Fig. 9. Diagram explaining the phenomena of Hemiopia and of crossed Amblyopia according to Charcot.

T, semi-decussation in the chiasma; TQ, decussation behind the corpora geniculata; a′, b, fibres which do not decussate in the chiasma; b′, a, fibres decussating in the chiasma.

b′, a′, Fibres proceeding from the right eye meeting at a point, LOG, in the left hemisphere. LOD, right hemisphere.

K, lesion of the left optic tract causing right lateral Hemiopia. LOG, a lesion at this point would cause right crossed Amblyopia.

T, lesion causing temporal Hemiopia. N, N, lesion causing nasal Hemiopia in either eye.

From the point of view of this theory, it is evident that a lesion situated at the point K, so as to interrupt the course of the fibres of the left optic tract (both those (b′) which decussate in the chiasma, and those (b) which do not decussate there), will affect the left half of each retina (G, G), or, in other words, will cause either diminution or complete suppression of the entire visual field on the right side (right lateral hemiopia).

Left lateral hemiopia would supervene in the same way in consequence of a lesion seriously damaging the optic tract on the right side.

It certainly is not uncommon to find such lateral hemiopia, as the theory indicates, resulting from disease involving the optic tract on either side, in any part of its course between the chiasma and the corpora geniculata. A tumour may originate in some part of the tract itself; or the tract may become involved secondarily, or pressed upon, by a tumour or a hæmorrhage occurring in the crus or the posterior tubercle (pulvinar) of the thalamus, or by a tumour originating in the inner and under part of the temporal lobe.

If the individual, affected in either of the ways above mentioned, chanced to be one in whom a total decussation of the optic fibres had taken place at the chiasma, then the symptom produced would be crossed amblyopia rather than lateral hemiopia.

A lesion situated in or rather involving the anterior median portion of the chiasma (T)—and such a lesion is most likely to be a tumour—would produce the same effects whether there is a total or a semi-decussation of fibres in the chiasma. The lesion would involve in each case decussating optic fibres (a, b′) and would thus paralyse the inner half of the retina on each side and consequently give rise to what is termed temporal hemiopia.

Where semi-decussation exists, and an injurious amount of pressure operates upon either outer angle of the chiasma (N, N), the effect would be the paralysis of the fibres emanating from the outer half of the retina, and consequently the suppression of the inner part of the visual field of the corresponding eye—that is the production of so-called nasal hemiopia. Knapp has recorded a typical example of this kind of hemiopia.

In the case of a complete decussation existing, a lesion in the same situation (N) would be more likely to produce a lateral hemiopia of the opposite side, as may easily be understood by reference to the figure. Thus, supposing the lesion to press upon the right side of the chiasma (N), it might paralyse the fibres coming from the outer half of the retina on the same side, as well as those from the inner half of the opposite retina (after decussation), thus giving rise to a left lateral hemiopia. I cannot refer to any such case, but future observers must not lose sight of the possibility of such a mode of production of lateral hemiopia. Still, its production by such a lesion might be denied by the advocates of the semi-decussation theory, seeing that, in accordance with their views, a lesion situated only a line or two further back, so as to involve the whole thickness of the first part of the tract, would also produce left lateral hemiopia.

So far as the original question is concerned, that is, whether the decussation of the optic fibres at the chiasma is a total or a semi-decussation, it will be seen that this is one which must ultimately be settled by two kinds of evidence—(1) by the accumulation of pathological evidence derived from cases of congenital blindness of one eye (or if not congenital by a unilateral blindness which has at least lasted several years) in which observation has been made as to whether or not the opposite optic tract has undergone an atrophy corresponding with that of the optic nerve on the blind side; and (2) by clinico-pathological evidence as to the effect produced during life in each case of marked disease of one of the optic tracts—lateral hemiopia being the result in accordance with the semi-decussation theory, whilst crossed amblyopia would occur if the decussation had been complete. This is really the only distinct issue that presents itself to the clinical observer.

It seems highly probable that there is truth on both sides of the question. Probably semi-decussation is the most common arrangement; but we want more information as to the degree of frequency with which a total decussation occurs. It seems clear from Fig. B that even an almost complete absence of decussation occurs in some cases, and I would suggest that this is even to be expected and looked for in such rare cases as those made known by the researches of Flechsig, where there is an almost complete absence of decussation of motor fibres in the anterior pyramids of the medulla—cases in which a motor paralysis of cerebral origin would occur on the same side as the lesion, rather than on the opposite side of the body. I have elsewhere indicated the possibility ('The Brain as an Organ of Mind,' p. 478) that in the vertebrate series the crossed motor relation between the brain and the body has been a necessary consequence of the decussation of the optic nerves, a relation which first becomes established in fishes. Both these crossed relations seem to be wanting throughout the invertebrata.

We must now turn to another question about lateral hemiopia which has given rise to much discussion. It is natural to ask, whether the lateral hemiopia that develops as a consequence of disease of the optic tract, will also result from a lesion which involves the optic fibres beyond the corpora geniculata, that is, in their deep intra-cerebral course. As before stated, this was the view put forward by von Graefe in 1860, and which, until recently, has been very commonly adopted. The clinical interpretation of this view is expressed by the dictum that, unilateral lesions of the cerebral hemispheres which affect sight produce lateral hemiopia and not crossed amblyopia.

There can be no doubt that this dictum, if true at all, is true only in part. This is shown by the facts already adduced concerning cerebral hemianæsthesia and its associated crossed amblyopia, caused by circumscribed lesions affecting the posterior third of the hinder division of the internal capsule. A correlated fact has been revealed by the earlier as well as the later researches of Ferrier ('Proc. of Royal Soc.,' 1884, Jan. 24, p. 222), who found that destruction of the angular gyrus on one side caused, not lateral hemiopia, but blindness of the opposite eye for a short period; whilst simultaneous destruction of both angular gyri was found to produce total blindness for a time not exceeding three days.

Stopping for a while here, it will be well to explain the notion which has been advanced by Charcot to reconcile these latter facts with that previously mentioned, that disease of the optic tract itself commonly produces lateral hemiopia. He puts forward the hypothesis that those outer optic fibres (Fig. 9, b, a′) which pass through the chiasma without decussating, cross one another posteriorly in the corpora quadrigemina (TO), and thence proceed to the cortex of the opposite hemisphere. This is confessedly a mere hypothesis; but clearly, if any such arrangement really exists, it would place the cortex of the cerebral hemispheres in the same kind of relation with the optic nerves as if there had been a total decussation at the chiasma. This view, that in at least the larger number of persons, half the decussation of the optic fibres takes place at the chiasma and the other half further back in the corpora quadrigemina, permits of the explanation of a great number of well-attested clinical facts.

If this view of Charcot in reference to the complementary decussation of the optic fibres in the corpora quadrigemina be correct, the following results should follow from lesions in these bodies alone, or in concert with the contiguous geniculate bodies, as may easily be understood by reference to Fig. 9:—

(*a*).—Lesions restricted to one of the anterior quadrigeminal bodies should produce nasal hemiopia; though, by some curious oversight, it is stated in Charcot's work (*loc. cit.* p. 119) that the result should be the production of crossed amblyopia.

(*b*).—If one anterior quadrigeminal body together with the external geniculate body of the same side were seriously damaged or cut off from their blood supply (as may happen, for instance, in a case of thrombosis of the posterior cerebral artery, which supplies these parts), the result would be the production of a crossed amblyopia, plus nasal hemiopia on the side affected. (The same lesion occurring in a person in whom the decussation of the optic fibres had chanced to be total at the optic chiasma, would give rise simply to crossed amblyopia.)

(*c*).—Lesions implicating the two anterior quadrigeminal bodies as well as the geniculate bodies on each side, should produce total blindness. This may happen as a rare event either from softening or from the growth of a tumour in this situation. Many years ago I saw a case of complete blindness caused by softening "almost limited to the anterior quadrigeminal bodies"; I cannot now say, as a matter of fact, that the softening did actually involve the external geniculate bodies as well, though it would seem most probable that this was the case.

Charcot thinks that lateral hemiopia does not occur at all as a direct result of lesions of the cerebral hemispheres. His words are :—"I do not believe that there exists at present a single observation proving satisfactorily that lateral hemiopia has originated in consequence of an intra-cerebral lesion, without there being any implication of the optic-tracts."

Confessedly this question is one beset with difficulties in regard to interpretation, and possibly Charcot's scepticism is here carried too far. Gowers still adheres to the opinion of von Græfe on this subject. He refers, with much confidence, to a case recorded by Baumgarten in which a hæmorrhage about the size of a walnut beneath the occipital convolutions seemed to be the only cause of a lateral hemiopia which had existed during life. He adds :—"The symptom has been due to tumours in this situation in many recorded cases, and in one which has come under the writer's notice." Ferrier, however, comes to an opposite conclusion ('Brain,' Vol. II, p. 97). He says :—"The cases on which the relation between hemiopia and lesion of the occipital lobe is founded are far from satisfactory. In Pooley's case there was also a lesion in the optic thalamus; in Herschberg's there was a tumour reaching to the optic thalamus; Wernicke's was a case of multiple lesion, both cortical and medullary; in Baumgarten's there was also lesion of the optic thalamus; and Nothnagel's own case was one of diffuse and multiple lesion in both hemispheres."

Some of Ferrier's most recent researches seem to harmonize with these and other clinical data. He obtained the following remarkable results by experimenting with monkeys:—

(1).—Destruction of one occipital lobe and one angular gyrus entails amblyopia of the opposite eye and loss of sight in both eyes towards the side opposite the lesion. It is doubtful whether the hemiopia is quite symmetrical. The visual disorder is temporary, the animal partially recovering in a week.

(2).—If in an animal which had recovered sight after destruction of the left angular gyrus, the right angular gyrus and occipital lobe were destroyed, the result was temporary left hemiopia.

(3).—If in an animal in which the occipital lobes had been removed without symptoms, the left angular gyrus was destroyed, the result was transient loss of vision in the right eye.

(4).—Destruction of both angular gyri and both occipital lobes caused total and permanent blindness.

Ferrier's conclusion from these experiments is that "there is a two-fold relation between the eyes and the cortical visual centres; one mainly crossed—the central portion of the retina being bilaterally represented—by the angular gyrus; the other bilateral (the corresponding side of both retinæ being represented—by the occipital lobe, not alone, but in conjunction with the angular gyrus). ('Brain,' vol. iii.)

It is important to bear in mind that one angular gyrus and one occipital lobe suffice, after the lapse of a few days from the date of the lesion, for vision with both eyes. This is quite in accordance with clinical facts, long ago recorded, in which the most extensive lesions in one hemisphere have left the sight of both eyes intact.

Again, it is important to bear in mind that destruction of one angular gyrus, causes a temporary crossed amblyopia; whilst destruction of the angular gyrus and of the occipital lobe, causes an opposite lateral hemiopia, in monkeys.

On the other hand, the cerebral hemianæsthesia occurring in man from structural lesions in the posterior third of the hinder division of the internal capsule, occasions a crossed amblyopia which is very much more lasting. What is the cause of this difference? Similarly, the lateral hemiopia which seems to result from some lesions in the occipital lobe of man, would appear to be a much more durable phenomenon than that produced by experimental lesions in this region in monkeys. This is another fact difficult to understand.

The lateral hemiopia that occurs from disease of the optic tract may, in some cases, be diagnosed from that resulting from lesions in the occipito-angular region of one of the hemispheres. Ferrier directs attention to the following points of difference between them :—

(*a*).—In the hemiopia due to pressure on the optic tract, the defect of vision is absolutely unilateral, because each optic tract derives its fibres precisely from the corresponding halves of both retinæ. [This, however, as we have seen (p. 137), is only true for some men.]

(*b*).—The hemiopia is probably of hemispheric origin when central vision is retained for some degrees on all sides of the point of fixation. (This is due to the fact that each angular gyrus has a bilateral relation with the centre of the retina, so that, in spite of the existence of disease in one visual centre, it is still possible for both eyes to possess central vision through the sound angular gyrus. Consequently, with a lesion in one occipital region, lateral hemiopia may be present towards the opposite side, but with retention of central vision in both eyes.)

(*c*).—Rapid atrophy of the optic discs occurring in association with hemiopia, would also be in favour of such hemiopia being due to pressure on the optic tract rather than to disease of the occipito-angular region of the hemisphere.

(4). *Diplopia.*—Double vision is a defect apt to be produced by paralysis of either one of the straight or oblique muscles of the eyeball, but is most frequently occasioned by a loss of the proper balance of power between the external and the internal recti muscles of the eyeball. This may be a result of a commencing paralysis or paresis of the external rectus muscle, and the diplopia is commonly most marked and distressing when the deviation of the optic axis from the normal is only slight, because the nearer the image of the squinting eye is to the yellow spot, the more definite will it be, and consequently the more difficult to distinguish from that of the normally directed eye.

Diplopia may, in the first place, be associated with convergent Strabismus, and this is due to three principal causes, to each of which brief reference will be made.

(a). Paralytic convergent strabismus is due to paralysis or paresis of the sixth nerve. Where there is actual paralysis, the defect is easily to be detected, and the diplopia speedily ceases to be troublesome, owing to the image in the squinting eye falling upon a part of the retina remote from the yellow spot, so that its lack of distinctness soon enables the patient to disregard it. In cases where there is only paresis of one external rectus, it is often impossible from mere inspection alone to say which is the eye at fault. A discrimination may be effected, however, and the paresis made manifest by an observation of the 'secondary deviation' of the sound eye. The measures to be adopted are stated almost in the words of Brudenell Carter (Quain's, 'Dictionary,' p. 1151). The observer should stand in front of the seated patient, and should hold before him, in the middle line, at the usual reading distance, some small object, which he is directed to look at steadily. By his own hand or by a piece of ground glass the observer should then cut off the view of the object first from one of the patient's eyes and then from the other, watching their movements as he does so. When the object is concealed from the squinting eye, the other eye, being already rightly directed, will remain stationary to continue the act of seeing, and the squinting eye will also remain stationary; but, when the object is concealed from the working eye, the other, or squinting eye, being wrongly directed, and not receiving the image of the object upon its yellow spot, will make a slight outward movement in order to take up correct fixation. At the same moment the working eye, behind the obstruction, will execute an inward movement of somewhat greater amplitude than the outward movement of its fellow.

During the first part of this examination, in which the squinting eye is covered, there is no movement because the unaffected eye is receiving a naturally clear and sharp image. When, however, this unaffected eye happens to be covered, the squinting eye receives the image of the object upon a point of its retina internal to its yellow spot, and sees it only indistinctly. The squinting eye, therefore, makes an excursion outwards, sufficient in amount to bring the image of the object upon its yellow spot, and to enable it to see better; but the motor impulse by which the necessary movement of the external rectus is called forth is conveyed at the same time to the internal rectus of the unaffected eye, as a result of the habitual association of the two eyes and of their muscles in the act of looking towards one side; and the sound muscle under a given motor impulse, contracts more vigorously than the weakened one. The result is that the excursion inwards of the working eye is larger than the excursion outwards of the squinting one; and in this way the fact of paresis of the external rectus of this latter eye is rendered manifest, by what is termed the 'secondary deviation' of the working eye.

(b) Hypermetropic convergent strabismus is due to the excessive development of the two internal recti which inevitably occurs in flat-eyed persons. This is a common cause of diplopia in children. As B. Carter puts it,—" The flat formation of the eye requires for acute vision of near objects, a strenuous accommodation effort; and this by the intimate association which exists between the nerve centres governing the accommodation muscles, and those governing the interni, produces a corresponding effort of convergence. As soon as a child who is born with flat eyes begins to take careful notice of near things, his accommodation muscles and his internal recti are both called into frequent and energetic exercise; and the consequence is that the interni become excessively developed in relation to their antagonists, the externi, so that the normal or resting position of the eyes, instead of being one of parallelism, becomes one of convergence. The result of this is that the child would receive double images of equal intensity, of all objects situated either nearer to him or farther from him, than the point at which the convergent optic axes would meet if prolonged. Let us suppose that this point is one foot distant from the eyes; and that the child wishes to look at an object which is eighteen inches distant. He cannot do this with both eyes; because the externi are unable to overcome their more powerful antagonists. If, however, he combines the right externus with the left internus, as in the act of looking to the right with both eyes, he becomes able to fix the object correctly with his right eye; and if he combines the left externus with the right internus, as in the action of looking to the left with both eyes, he becomes able to fix the object correctly with his left eye. But as, in either case, both eyes start from a position not of parallelism but of convergence, the effort which carries the right eye from its convergent state to the middle of its palpebral fissure will carry the left from its convergent state to one of much greater convergence, and *vice versâ* with the left eye; so that while one eye is directed to the object of vision the other is rolled far inwards. In this way the image is received upon the yellow spot of the working eye, and upon so peripheral a portion of the retina of the squinting eye that it is easily neglected by the consciousness, and ceases to be a source of confusion and embarrassment."

In a case of this kind, during a state of rest the eyes are equally convergent; but, as soon as any object is looked at, one eye fixes this object and the other eye rolls inwards. If the eyes are similar in regard to their flatness and the state of their muscles, it is important to remember that sometimes one will be the squinting eye and sometimes the other. The squint is then said to be 'alternating.' Generally, however, especially after a time, some amount of difference becomes established in regard to the two eyes, so that it becomes easier for the person to work with one eye than with the other. This eye is then used in preference to, and gradually supersedes, its fellow—the one becomes always the working, while the other remains the squinting eye. The squint is then said to be 'permanent.'

(c) Myopic convergent Strabismus.—The convergent squint of short-sighted people is quite a rare affection. It is due to the same kind of inequality between the internal and the external recti which exists in the variety last referred to. When spectacles have not been worn by short sighted persons, the externi which produce the approximate parallelism of the optic axes necessary for distant vision, can have been exercised only in a very imperfect manner. The interni, on the other hand, have been over exercised in the constant production of convergence for the vision of near objects. In such cases the eyes are usually equally convergent, such a pos ition giving single vision of near objects; in more distant vision a certain amount of diplopia occurs—it is however scarcely complained of, on account of the obscurity of the images in all distant vision in such patients.

Divergent strabismus productive of diplopia occurs from paralysis or paresis of the branch of the third nerve which supplies the internal rectus muscle. The extent of the divergence is, after a time, increased by secondary shortening of the unantagonised muscle. This and other forms of diplopia occurring from paralysis of branches of the third nerve will be better referred to under that head, since the exact nature of the diplopia constitutes an important part of the signs indicative of paralysis of this or that ocular muscle (see Part III. Paralyses due to Lesions of Cranial Nerves).

A description of the diplopia associated with paralysis of the fourth nerve is deferred for a similar reason.

The diplopia associated with disease of the sixth nerve has been referred to here because it is essential that it should not be confounded with the other two forms of convergent squint (viz., the hypermetropic and the myopic forms) each of which requires the especial care of an oculist, though they are of no importance whatever in regard to the localisation of brain disease.

It is, of course, highly important that the student should be able to distinguish the several forms of diplopia associated with paralysis of the sixth, of the different branches of the third, and of the fourth nerves, because the recognition of these several forms of paralysis may help us, at times, to localise lesions in the brain.

XI. PARALYSES OF CRANIAL NERVES.

Paralysis of one or other of the Cranial Nerves, or combinations of such paralyses, are often of the greatest importance in helping to localise lesions within the cranium.

In order to utilise such signs, the course and relations of each nerve within the cranium have to be borne in mind—that is, its relations from the point of attachment to the brain, to the point where it passes through this or that cranial foramen. The course of the several nerve roots within the brain substance must also not be lost sight of, as well as the several relations of these and of their nuclei with one another. All these are purely anatomical details, the knowledge of which, however, will often prove of the greatest service for the determination of a regional diagnosis.

Nothing more can be done here than to insist upon the importance of such signs. Elsewhere the signs indicative of paralysis of each cranial nerve will be given separately (Part III.); whilst, in speaking of the indications of disease in the crus, in the pons Varolii, and in the bulb, some of the combinations in which such paralyses are prone to present themselves will be referred to. In reference to disease in other brain regions, this important subject will also be considered. Still combinations of such paralyses will constantly occur in practice, in which the question will have to be considered whether they can be produced by a single lesion, or whether they are the results of two or even more separate lesions, related to one another merely by some common under-lying condition, such as syphilis or tuberculosis, or by the existence of some form of cerebro-spinal sclerosis, or nuclear atrophic disease.

Finally, it is often of the highest importance to determine whether disease affects some particular nerve in its intra-cranial or in its extra-cranial course. This is a question the consideration of which comes up more especially in regard to the facial nerve.

XII. HEMIANÆSTHESIA—CEREBRAL AND MESOCEPHALIC.

This is a sign of considerable importance in regional diagnosis because it has a very distinct localising value.

Hemianæsthesia presents itself with two different degrees of completeness, from the point of view of range. The most complete form is known as cerebral hemianæsthesia, whilst the less complete form is distinguished as mesocephalic hemianæsthesia. The former set of signs has a precise regional value; the latter is again divisible into two sub-varieties and these have localising values which are scarcely if at all less definite, if we pay proper

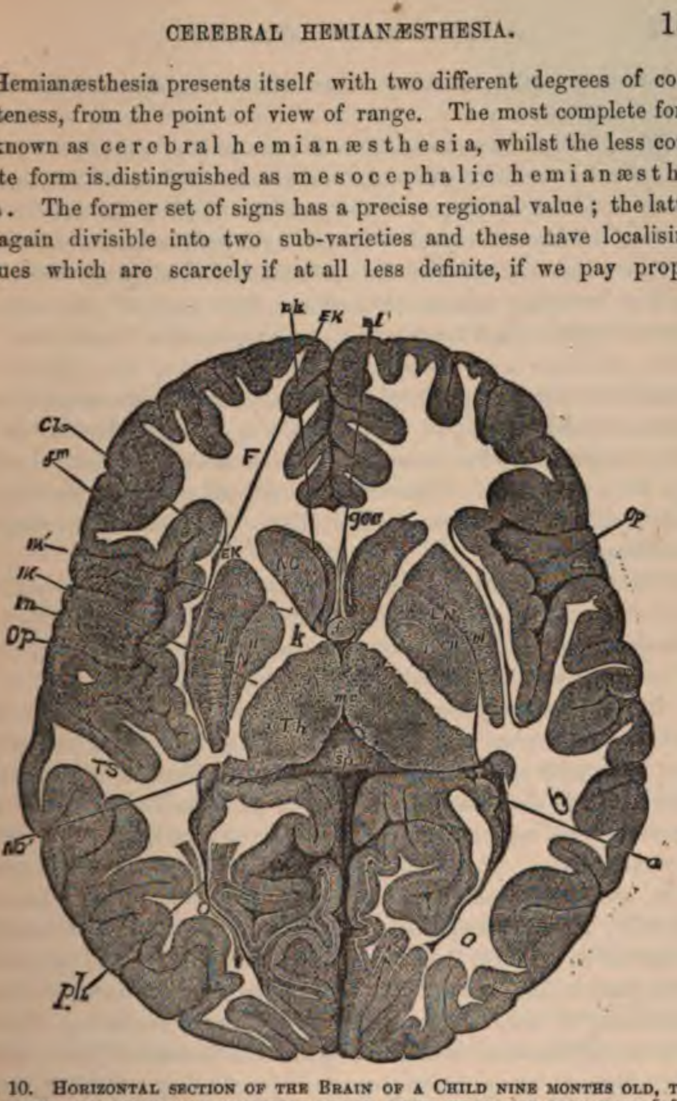

Fig. 10. Horizontal section of the Brain of a Child nine months old, the right side being at a somewhat lower level than the left half [after Flechsig].*

F, frontal; TS, temporo-sphenoidal; and O, occipital lobes. Op, operculum. In, Island of Reil. Cls, claustrum. f''', third frontal convolution. Th, thalamus, NC, caudate nucleus NC′, tail of caudate nucleus. LN, lenticular nucleus. 1,11, 111, first, second, and third divisions of the lenticular nucleus. EK, external capsule. IK, posterior division; IK′, anterior division; K, knee of the internal capsule. ab, ph, anterior and posterior horns of the lateral ventricles. gcc, knee of the corpus callosum. sp, splenium. mc, middle commissure, f, fornix. sl, septum lucidum. a, cornu Ammonis.

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* To obtain this view, Flechsig makes a horizontal section of the brain just above and parallel with the Sylvian Fissure.

attention to other collateral signs with which they are apt to be associated.

(a). *Cerebral Hemianæsthesia.*—It has now been conclusively proved by clinical as well as by experimental evidence that cerebral hemianæsthesia is caused by lesions of the posterior third of the hinder segment of the internal capsule—viz., that part of the foot of the corona radiata which lies between the optic thalamus and the posterior part of the lenticular nucleus (Fig. 10). This part of the brain is supplied with blood by the lenticulo-optic branches of the middle cerebral. Hemianæsthesia of the type we are considering may be produced by the rupture of these vessels; or by their occlusion, leading to softening of the posterior third of the internal capsule. When caused in this manner the hemianæsthesia is apt to be permanent. Tumours growing in this region of the brain, as a rule, produce much less decided effects than either hæmorrhage or softening.

Either of these pathological changes when occurring near, though not actually involving the posterior third of the hinder segment of the internal capsule, may, by causing undue pressure upon this region for a time, lead to a less durable form of cerebral hemianæsthesia.

Before describing the condition itself, it is important to mention that a state in every way similar, except that it is apt to be fluctuating in severity and altogether irregular in its duration, is produced at times in girls and women, as a result of functional disturbance—presumably in the region above mentioned though independent of all discoverable lesion. This state may occur alone or it may be associated with such remarkable convulsions as have been already described (p. 90); the whole condition then corresponds with what Charcot has described as Hystero-Epilepsy. It is important to bear in mind, however, that in some cases no distinct hysterial manifestations, or other symptoms of such a state, are to be found coexisting with an apparently functional hemianæsthesia. It will scarcely be contended, even by those who are most prone to arrive at a diagnosis of 'hysteria' on slight provocation, that functional disturbance cannot occur in this or that region of the nervous system apart from hysteria.

When due to hæmorrhage or vascular occlusion this form of hemianæsthesia may present itself in association with a more or less complete hemiplegia of the same side of the body (with or without an apoplectic mode of onset). The anæsthesia is strictly unilateral, or it may pass very slightly beyond the middle line, both in front and behind. It affects limbs, trunk, side of the face and head, all in the same manner—involving the mucous membranes as well as the skin. All modes of sensibility are alike implicated; there is inability to appreciate touch, pain, or differences of temperature. The loss of sensibility may also affect the deeper textures of the limbs or other parts of the body. When it exists this is particularly noticeable in the muscles, which remain painless under the influence of the strongest Faradic currents, although they are caused thereby to contract freely.

Variations naturally exist in regard to the completeness and the degree of hemianæsthesia of the limbs and trunk, when the condition is due to an organic lesion; but precisely similar variations may also be met with when the disease is of functional origin.

Again, it is not in all cases of either category that the special senses are involved; and when there is an absence of such implication, we lack the distinguishing characters which specially differentiate this cerebral form from mesocephalic hemianæsthesia.

But in the complete and typical forms of cerebral hemianæsthesia, all the special senses are affected. Not only is there loss of taste on the corresponding half of the tongue, and loss or impairment of hearing on the same side; there is, in addition, loss or impairment of vision and of smell on the affected side. The peculiarities connected with the sense of vision apt to be met with in these cases, need not now detain us, since they have been already described under the head Amblyopia (p. 134).

(b). *Mesocephalic Hemianæsthesia.*—This form of hemianæsthesia, may be produced either by disease of the cerebral peduncle, or by disease in the pons Varolii. Both varieties are distinguished from cerebral hemianæsthesia by the absence of implication of smell and sight—a negative sign easily explicable on anatomical grounds.

Lack of sensibility of the mucous membrane of the nostril on the affected side to pungent vapours, such as ammonia, must not be confounded with loss of smell. Again, there may be some slight visual trouble on the affected side due to disturbance of accommodation in these cases, although vision is interfered with in no other way, and the functional activity of the optic nerve is not at all impaired.

The special senses of taste and hearing are implicated on the same side as the general sensibility of the body and limbs—viz., on the side opposite to that on which the lesion exists.

It is desirable, however, that in future cases special attention should be given to the degree and kind of implication of hearing in disease of the cerebral peduncle—looking to the positively expressed opinion of Meynert that "no extensive immediate connection of the auditory nerve with the cerebral lobes exists." He contends that the connection is brought about indirectly through the Cerebellum (Stricker's 'Histology,' Vol. I., p. 500). If that be really true, then lesions of the upper half of the pons, and still more of the cerebral peduncle, ought not to cause impairment of hearing. If Meynert's view be correct, it would seem that the auditory fibres could only reach the posterior third of the internal capsule by way of the upper cerebellar peduncles.

It is well to state that the special senses of taste and hearing are not necessarily involved in all cases of mesocephalic hemianæsthesia. They occasionally escape, as I have found.

Both in the peduncle and in the pons the sensory fibres seem to be aggregated together in a comparatively limited area distinct from, though contiguous to, the main motor tracts. In either region, tumours, hæmorrhages, or more rarely softening may occur, which may involve the sensory tracts alone (this is very rare), or the sensory and the motor tracts in varying degrees.

The diagnosis of disease in either of these localities must of course always be dependent upon the particular grouping of signs and symptoms presented. These are given elsewhere (see Paralyses due to Lesions in the Crus Cerebri, and Paralyses due to Lesions in the Pons).

It has now been ascertained that the upper and outer layers are the parts of the peduncle the destruction of which, or the interference with whose functions by disease, leads to the production of hemianæsthesia. Again, it has been ascertained that in the pons the sensory fibres occupy a similar situation, so that hemianæsthesia is produced by lesions in this part which involve the outer regions of the upper (or upper and middle) strata of the pons.

XIII. DIFFERENCES IN TEMPERATURE ON THE TWO SIDES OF THE BODY—PARALYSED AND NON-PARALYSED.

It is very common, for some days, weeks, or months from the commencement of a hemiplegia, to find that there is a distinct difference between the temperature of the limbs on the paralysed and the non-paralysed side of the body. The temperature taken in the axilla on the paralysed side is commonly from ·5°-2° F. higher than it is in the same situation on the non-paralysed side. (But where hemiplegia has existed for some years it is apt to be otherwise; the paralysed limbs may be then distinctly colder than their fellows, and the skin may be of a bluish red colour. These are remote effects consequent upon prolonged disease.)

And, as we have already pointed out, this difference in temperature is appreciable from the first. Even in the apoplectic stage, when it exists and is due to a unilateral cerebral lesion, such a difference of temperature may commonly be noted. It is more marked in the upper than in the lower extremities; and as Lépine points out, the difference between the former is often easily recognizable by feeling the palms of the hands.

It is commonly believed that the higher temperature on the paralysed side is due to an elevation there above the normal, varying within the limits just mentioned; and that the normal axillary temperature is to be found on the opposite or non-paralysed side of the body. Observations, however, made in many cases, and extending in each over days or weeks, have shown me that this is generally not the case. I have found, on the contrary, that the axillary temperature on the non-paralysed side (that is, on the same side as the cerebral lesion) is often distinctly subnormal, whilst that on the paralysed side is not above the normal—as may be seen by comparing it with the temperature of the mouth taken simultaneously. In such cases it would seem that the brain lesion exercises an irritative influence upon the chief vaso-motor centre situated on the same side of the brain (in the medulla), and that this chief vaso-motor centre is in relation with the blood vessels of the same side of the body. Is this latter supposition correct or not? Is there evidence to show that the supreme vaso-motor centre in the right half of the medulla can exercise any influence on the calibre of the blood vessels (and consequently on the temperature) of the left side of the body? Or, is its controlling influence limited solely to the right side of the body, as I now suppose? In other words, is the influence of these supreme vaso-motor centres direct or crossed?

I was formerly under the impression, commonly entertained, that the difference of temperature on the two sides of the body in a case of hemiplegia, was attributable to the existence of a temperature actually higher than the normal on the paralysed side; whilst now I know that in many cases, at least, the difference is rather due to a depression below the normal on the non-paralysed side or side of lesion. The former condition was supposed to be in relation with the larger radial pulse (and, as it was thought, probable general relaxation of vessels) commonly met with in recent cases of hemiplegia on the paralysed side. Yet this, too, may have been an erroneous interpretation. The apparently larger pulse on the paralysed side, may be only relatively large, when compared with the actually diminished pulse on the side of the cerebral lesion.

Seeing that the facts are as I have just stated them, it is essential that they should be spoken of in a different way. Instead of saying that the temperature is higher on the paralysed side, it would be better to say that the temperature is lower or sub-normal on the side of lesion.

This subject stands in need of further investigation, especially as it is one to which extremely few references are to be found in medical literature. Bourneville supplies no facts which throw any light upon the special aspect of the question to which we have just been referring.

For the present the bearing which these differences of temperature have upon regional diagnosis is very slight. We can only speak in somewhat general terms upon the subject, and say that in lesions of many parts of the cortex, at least, the difference of temperature in the two axillæ rarely exceeds 1° F.; whilst in lesions in the pons Varolii the difference not unfrequently amounts to 2° F., or even more.

Lesions in the corpora striata seldom give rise to a difference of more than 1° F.; but with lesions in and just outside the thalamus I have found a difference of 1·5° F. on several occasions.

These differences between the temperature of the paralysed and the non-paralysed side of the body are often less in the morning, and become decidedly more marked towards evening. Here, for instance, is an example of such a daily variation, observed in a man incompletely paralysed on the left side, just fourteen days from the onset of his illness (which was subsequently found to be due to a hæmorrhage into the right optic thalamus) :—

	L.	R.
11.A.M.	97·5	97·2
3.P.M.	98°	97·8
7.P.M.	98·8	97·6

Some observations made during the last three or four years have, moreover, convinced me that there are exceptions to the general rule that the temperature on the non-paralysed side is lower than that on the paralysed side. I found for instance in the case of a patient under my care about two years ago in University College Hospital, that the temperature for the first four or five days after the commencement of the paralysis (which was initiated without loss of consciousness) was lower rather than higher on the paralysed side; though after this time, it continued for some weeks to be slightly lower on the non-paralysed side, though rarely more than by ·5° F. Again, in another case of left hemiplegia supervening without loss of consciousness, and admitted to the same hospital within half an hour from its onset, the temperatures were regularly taken on the two sides of the body during the whole three months that the man remained under observation, with the result that during the first two and a half months the temperature remained lower on the paralysed side—being there sub-normal and from 0·5-1° lower than it was on the non-paralysed side (the temperatures having always been taken at 11 a.m.). After two and a half months the recovery of power in the paralysed limbs became more distinct, and then after some oscillations the temperature speedily equalized itself on the two sides. Judging from the patient's condition as a whole, I was disposed to think that the lesion in this case was a hæmorrhage which interfered for some time with the functions of the posterior third as well as the anterior part of the posterior division of the internal capsule. As to the cause of these exceptional conditions I am not prepared to offer any opinion.

Again, quite recently, in a case of apoplexy due to cerebral hæmorrhage, I have noted a remarkable reversal of the temperature of the two sides of the body, taking place a few hours before death, whilst the general body temperature was rapidly rising, as it often does in fatal cases of this type. The diligent and careful manner in which the temperatures were taken by my house-physician, Mr. Wm. Carr, brought out some interesting results which I shall briefly attempt to explain. The patient was an engine driver of the Scotch express, admitted to University College Hospital under my care on the evening of March 3, 1885, in a partially comatose condition and hemiplegic on the left side. During the first two days his condition underwent some slight improvement; then he became more comatose apparently from an increase of the hæmorrhage: the comatose condition gradually became more profound during two more days. At the expiration of this time there was general resolution of all limbs, and disappearance of all reflexes on both sides of the body. Within a few hours of this period, that is some hours after midnight on the 8th, when the final general rise of temperature had well commenced, the temperatures on the two sides of the body had become reversed—being by this time higher on the side of lesion—and so they continued during the remaining eight hours of the patient's life. Yet during the previous four days the records had shown on all occasions except the first (when they were registered as equal), a lower temperature on the side of lesion. Some of the temperatures taken are here subjoined :—

March	*Left.*	*Right.*	*Rectum.*
3rd	97°	97°	98°
4th	97·8	97·4	99°
5th	98·2	97·6	98·8

March	*Left.*	*Right.*	*Rectum.*
6th	100·2	100°	101·2
7th	100·4	99·4	101·4
8th 4 A.M.	102·4	103°	104°
,, 6 ,,	102·2	103·2	104°
,, 8 ,,	103·4	104·4	105°
,, 10 ,,	103·4	105°	105·6
,, noon	105·6	106·2	106·8

At the autopsy, I found much evidence of pressure over the parietal and frontal regions of both hemispheres—most marked, however, on the right side, where the convolutions were notably flattened and the sulci obliterated. There was a very large hæmorrhage into the substance of the right hemisphere, lying outside and also involving the lenticular nucleus and the thalamus. No extravasation into the ventricles and no lesion in any other part of the brain. There could be no doubt that a hæmorrhage of such an extent had not existed at the time of the patient's admission or for two days afterwards, when there was no profound loss of consciousness and nothing more than a condition of stupor existed. After that time, further bleeding evidently began to take place into the hemisphere, and with it the coma began to deepen. One effect of the pouring out of the increased amount of blood into the hemisphere must have been greatly to increase the pressure upon certain parts of the brain. Marked evidence of this was seen after death in the right fronto-parietal region, and only to a slightly less extent in the left fronto-parietal region.

We must here make a digression concerning the possible existence of 'heat centres' in the brain—that is centres which, aided by a special set of fibres issuing from them, exercise a restraining influence upon the chemical or metabolic changes taking place in the body, and consequently a restraining influence upon the amount of heat generated. Much evidence of a clinical, as well as of a physiological nature, has now been accumulated upon this subject, which has been recently reviewed in a careful manner by Dr. Hale White ('Guy's Hosp. Reps.,' Vol. XLII.). He adduces clinical reasons for supposing "that there is a calorific centre on the surface of the brain in the region of its middle third; somewhere, that is to say, in the neighbourhood of the fissure of Rolando." An examination of the physiological evidence shows also that this is the very part of the brain "where Hitzig, Wood, and others, have localised the heat centre in dogs." Looking to the great height to which the temperature of the body often rises when the supposed restraining influence of these centres is destroyed, some reason exists, as Dr. White points out, for supposing that "there is always a very strong inhibitory influence passing to the tissues to restrain their metabolism," or which, at all events, has an effect of this kind, to whatsoever other purpose such influence may minister.

Now, looking upon what has been said in the last paragraph as merely about the best working hypothesis, upon which to explain a large number of clinical and physiological facts, let us see whether the same hypothesis is capable of throwing any light upon the rise of temperature which occurred during the final hours of life in the above-cited case, and also upon the remarkable reversal of temperature that followed on the two sides of the body.

The final rise of temperature in this case was distinctly preceded for some hours by the coma becoming absolute, the paralysis becoming general, and by the complete disappearance of the knee jerk (which had previously been normal) on the *right* side. These results might be explained by the gradual increase of pressure within the cranium causing an abolition of function in the Rolandic region and other parts of the previously undamaged left hemisphere. As this pressure became more marked on the Rolandic zone of both hemispheres (owing either to the bleeding still continuing, or from an increasing congestion and œdema consequent upon the altered conditions of the cerebral circulation) the 'heat centres,' assumed to exist in this region, may have gradually become paralysed, so that the heat of the body progressively increased, as it did in this case during the last ten or twelve hours of life.

Assuming this to be a probable explanation of the final rise of temperature in this particular case, how are we to explain the reversal of the temperature on the two sides of the body which also occurred at this period? I am disposed to think it may be explained in this way. The original difference of temperature observed on the two sides of the body up to March 5, was of the ordinary hemiplegic type, being greater on the paralysed side, owing to its being depressed below the normal on the non-paralysed side, or side of lesion. This latter condition we assume to be a result of irritation affecting the great vaso-motor centre on the same side of the medulla oblongata. Even during March 6th and 7th, when a moderate febrile condition existed, the same kind of difference between the two sides was maintained, as is commonly the case. But, on the following day (8th), the conditions previously regulating the temperature of the two sides of the body seem to have become obscured by a much more potent cause, viz., paralysis of the 'heat centres,' owing to the great increase of pressure upon the Rolandic zones in which they are supposed to be situated. As was noted at the time of the autopsy, the pressure had evidently been much greater on the side of lesion than on the left side. There would be reason for believing, therefore, that the paralysis of the 'heat centres' was also more marked on the right side, and that this may have led to a greater generation of heat on the right side of the body—the difference being adequate to bring the temperature of the right side, in these final stages, above, rather than below, that of the left side of the body.

It can scarcely be supposed that the original lesion tore across the efferent fibres from the right 'heat centre.' If it had been so, there ought to have been a general and a special rise of the temperature soon after. Although we know nothing as to the course pursued by these efferent fibres, we may assume, therefore, that they had escaped injury, and that the subsequent signs of paralysis of these centres were due to the increasing pressure on the Rolandic regions of the hemispheres. If this explanation is to be accepted in regard to the general rise of temperature, then there certainly was reason for believing that the paralysis from pressure would be greater on the right than on the left half of the brain; and seeing that the previously lower temperature of the right side of the body now became, and continued to be, more elevated than that of the opposite side of the body, the natural inference would be, that the 'heat centres', like the vasomotor centres, have a direct rather than a crossed relation with the sides of the body.

Thus, the simple explanation of the reversal of the temperature of the two sides of the body towards the close of this case of apoplexy seems to be, that the more marked paralysis of the 'heat centre' on the side of lesion, more than sufficed to antagonize the previous effects due to irritation of the principal vaso-motor centre on the side of lesion.

The notion that the influence of the 'heat centres' upon the sides of the body is direct rather than crossed runs counter to views previously expressed, though not, I believe, to any definite facts. A precisely similar remark may also be made in reference to the conclusion here also drawn that the influence of the principal vaso-motor centre is direct rather than crossed. The two conclusions are in accord with one another, and there would be nothing contradictory in such an arrangement. If for certain reasons which I have elsewhere considered ('The Brain as an Organ of Mind,' 1880, pp. 478-480), a cross relation has been brought about between the sensory and motor strands having to do with the life of relation of vertebrate animals, it does not in the least follow, as a necessity, that any similar crossed relation would become established between the sensory and motor nervous strands having to do with the mere organic or vegetative life of such animals. I have indeed given reasons for supposing (*loc. cit.*, pp. 536 and 546) that the nervous channels conveying afferent impressions from viscera, and perhaps even those conveying olfactory impressions, do not decussate on their way to the cerebral cortex—or in other words that the centres for these impressions also have direct rather than crossed relations with the body.

XIV. DISTRIBUTION OF MOTOR PARALYSIS IN FACE, LIMBS, AND TRUNK.

It is now a commonly known and understood fact that disease or injury in one half of the brain which injuriously affects its motor channels or tracts, leads to motor paralysis of the opposite half of the body. This, then, is one of the most general or primary facts in regional diagnosis.

Anatomists and pathologists have alike shown that a considerable proportion of the motor channels proceeding from each hemisphere of the brain are accustomed to 'decussate' in the medulla on their way to the opposite lateral column of the spinal cord. This decussation of the motor channels in the medulla, affords a sufficient explanation of the crossed motor relation between the one hemisphere of the brain and the opposite half of the body.

On the other hand, clinico-pathological evidence has shown that, in certain rare and exceptional cases, the lesion in the brain has occurred on the same side as the paralysis of the body—so that in such individuals, at all events, the motor relations between the halves of the body and the sides of the brain are direct and not crossed. About twelve years ago this subject gave rise to much discussion, and when lecturing at that time, after adverting to the varying degrees of completeness of this decussation in some of the lower animals, I said (' Paralysis from Brain Disease,' 1875, p. 212) :—" But if the anatomical structure of the cord and medulla varies amongst vertebrate animals, is it not possible that vices in development may occasionally occur in the nervous system of man to such an extent that the accustomed decussation of the motor tracts does not take place ? " I further suggested that in any subsequent case of the kind, the tract of secondary degeneration through the peduncle, medulla, and cord should be especially looked to, in order to see what amount of truth there was in this suggested explanation. Since this date the developmental researches of Flechsig have been published, and by these lines of enquiry he has been enabled to show that variation does occasionally exist in the relative proportion of the decussating and the non-decussating fibres in the medulla, and that in certain rare cases the decussation, if not absent, is of the most trivial character. Supposing one of these very rare individuals to be the subject of a brain lesion damaging the motor tract, we should then have to do with one of those exceptionally rare cases, from a clinical point of view, in which paralysis of the body occurs on the side of the brain lesion.

A most important part of Flechsig's researches has been directed to tracing out the pyramidal system of fibres from the medulla downwards into the spinal cord, as well as upwards through the brain. He finds that they are an adjunct to the fundamental spinal tracts, and are here always developed at a later period than these latter. Their development coincides with that of the cerebral hemispheres. In the cerebral hemispheres, the pyramidal strands can be traced upwards to the convolutions bounding the fissure of Rolando; whilst in the spinal cord they can be traced downwards, partly in the anterior columns and partly in the lateral columns. It has been long known that, as a rule, the larger proportion of the fibres pertaining to the cerebral tract of one side pass into the opposite lateral column of the spinal cord, the remainder not decussating but passing down in the inner part of the anterior column of the same side. Flechsig's investigations have shown, however, that there are numerous important exceptions to this rule, and that the differences which present themselves can be ranged under two or three distinct types.

He says (' Die Leitungsbahnen im Gehirn und Rückenmark,' 1876, p. 272): —" The question is, can we look upon any fixed proportion of the pyramidal strands as the normal ? It is evident from the table that the variations are not mere casual exceptions, but that variability is rather the rule. We have two extremes between which there are a number of intermediate grades. The extremes are those in which the pyramids either pass entirely into the lateral columns, and therefore the anterior columns are reduced to O, or the latter contain 90 per cent. of the pyramidal strands and the former are reduced to a minimum. The commonest modification is that there are four pyramidal tracts. Yet this can scarcely be regarded as the rule as there are so many modifications. If we take the case in which neither of the anterior columns at the cervical enlargement sinks below three per cent. or rises above nine per cent. as approximately equivalent, we might regard this as the normal."

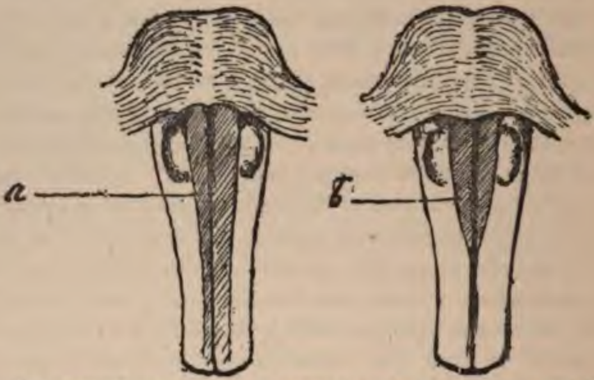

FIG. 11.—THE TWO EXTREME TYPES AS REGARDS DECUSSATION OF THE PYRAMIDS [from Charcot after Flechsig].

a, Almost complete absence of decussation, and consequent great predominance of the direct pyramidal tract.
b, Type of total decussation, in which the direct pyramidal tract is absent.

Flechsig reduces the different varieties to three types:—

The first and the most common (75 per cent.) is semi-decussation of both pyramids—each pyramid supplying a direct and a crossed bundle. In the great majority of cases the direct is much less important than the crossed fasiculus and is represented by from 3 to 9 p.c. of the pyramidal fibres, whereas the crossed fasiculus contains from 91 to 97 p.c. of these fibres.

In two cases, the one observed by Flechsig (out of sixty cases examined), the other by Pierret, the proportion above cited has been about reversed, that is the direct fasiculus has been represented by about 90 p.c. of the total number of pyramidal fibres on one side, while the crossed fasiculus has been represented by only 10 p.c. The number of fibres which decussate is here so insignificant that they need scarcely be taken into consideration. This constitutes the first extreme type above referred to (Fig. 11, a).

Many intermediate cases belonging to this first type are met with, which lie between the most common arrangement and that rare one just referred to.

The second type is that in which the decussation is total, or, in other words, that in which the direct fasiculi are absent. This constitutes the second extreme above illustrated (Fig. 11, b), and was met with in 11 p.c. of the subjects examined.

The third type is that in which there is semi-decussation of one pyramid with total decussation of the other. This asymmetrical arrangement was met with in 14 p.c. of the cases examined. Sometimes the semi-decussation occurs on the right and sometimes on the left side.

As Charcot points out, it is important to remember that asymmetry in the cord will result from asymmetry in the decussation; so that all cases of asymmetry in the cord are not necessarily of pathological origin.

The fact that merely one half of the body, or rather a part of one half of the body, is commonly affected in paralysis of cerebral origin is not difficult to explain. The two halves of the brain or cerebral hemispheres are in great part separate and distinct structures; lesions, therefore, are apt to occur in one or other of them, and consequently to paralyse one or other side of the body with various degrees of completeness. This is the reason why the typical form of paralysis of cerebral origin occurs so commonly as a hemiplegia. Directly we come to consider the effects of lesions in that part of the brain in which its two halves are fused into a close union, viz., the pons and the bulb, we find that the previous rule no longer holds good—that is, the motor paralyses which occur from disease is these parts is no longer strictly unilateral, but often more or less bilateral.

Our object here is to look to the various forms and degrees of paralysis from the point of view of a regional diagnosis, therefore, we must in the first place (1) specify what parts of the hemispheres form the starting points of motor incitations, and what are the routes followed by such incitations towards the medulla and spinal cord, on their way to the muscles. Subsequently (2) we must endeavour to ascertain in what way, or, to what extent, different degrees and modes of grouping of motor paralysis are indicative of a lesion in this or that region of the parts or tracts above indicated.

1. Seats of Origin and course pursued by Motor Incitations through the Brain. It is a truth so plain that it may almost be regarded as an axiom in nervous physiology that motor incitations always have their starting points in sensory cells or centres. It is true that in a simple 'reflex action' the incitation coming from the periphery by some afferent nerves, seems merely to pass through the sensory cells in order to rouse into activity the motor side of the reflex arc. It is none the less true that the gentle direct stimulation, by electricity or other means (if that were possible), of the sensory cell or cells on the ingoing side of any simple reflex nervous mechanism, would rouse the activity of the motor side of the reflex arc in the same manner. The same thing must hold good for more and more complex nervous centres. If there is reason to believe that the activity of motor centres is in any way guided or regulated by influences emanating from sensory centres, then there is all the more reason for supposing that the stimulation of such sensory centres would evoke the movements which are accustomed to be produced under their influence. This subject has been previously discussed (see p. 109), and the conclusion above drawn seems perfectly legitimate.

The question is now again referred to for obvious reasons. It has been definitely ascertained by Hitzig, Ferrier and others that a particular region of the cortex cerebri is excitable under electrical stimulation, and that definite movements can be evoked by stimulation of definite portions of this excitable region. It is known to all that for such and allied reasons, this excitable portion of the cortex is supposed to contain a congeries of 'motor centres.'*

Whilst gratefully accepting the experimental facts which have been demonstrated, I altogether demur to the interpretation. I do not believe that any motor centres exist in the cerebral cortex; on the contrary, I believe that what have been termed motor centres are in reality sensory centres of the Kinæsthetic type—and that for reasons which I have previously set forth (p. 113). The stimulation of such centres might be expected to produce the movements to which they were related; and, similarly, the destruction of such centres should deprive the animal of the possibility of producing such movements. But let us now define the situation of these centres, in which movements (that is, the ingoing impressions resulting from movements) are represented in the cerebral hemispheres.

The **excitable regions** in each cerebral hemisphere are similarly situated. They have been most precisely defined by experimental observations on the lower animals, but especially upon monkeys. Already, however, the natural experiments made upon man by this or that localised morbid process, is tending to show pretty conclusively that the distribution of the several centres in the human subject is essentially similar to what it is in monkeys.

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* After speaking of this excitable region of the cortex, Charcot says ('Localisation,' p. 193):—"I take the present opportunity of reminding you, incidentally, that the denomination, motor centres, does not imply in my mind any absolutely fixed physiological idea, but that by it I merely wish to designate in opposition to other areæ, those regions of the cerebral cortex, lesion of which occasions motor disorders in certain well-defined parts on the opposite side of the body."

The excitable region is situated mainly in the two great convolutions lying in front and behind the fissure of Rolando, that is in the ascending frontal and the ascending parietal convolutions; it also extends slightly backwards above, into the parietal lobule, and slightly forwards into the posterior extremity of each of the three frontal convolutions.

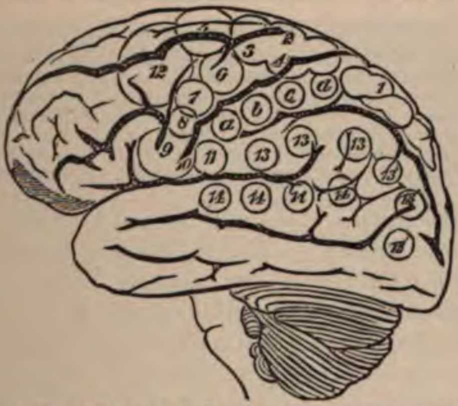

FIG. 12. LEFT HEMISPHERE OF THE CEREBRUM, SHOWING DIFFERENT CENTRES IN THE EXCITO-MOTOR AREA OF THE CORTEX GROUPED ROUND THE FISSURE OF ROLANDO [after Ferrier].

FIG. 12.

CENTRES FOR THE LOWER LIMB, WITH THE MOVEMENTS TO WHICH THEY ARE RELATED.

1, Postero-parietal lobule. Flexion of the foot at the ankle, occasionally combined with flexion of the thigh on the pelvis, and extension forward of the leg—as in the act of walking.

2, Upper extremity of the ascending frontal as well as part of the ascending parietal. Movements bringing the foot towards the middle line of the trunk, as if the animal were about to scratch its abdomen or lay hold of something in this position.

3, Upper part of ascending frontal. Movements of the tail.

Ferrier ('Localisation,' p. 65) says:—"It is a question by no means easy to answer *à priori*, how far the movements of the human leg can be compared with those of the leg-arm and foot-hand of the monkey; or what is the representative in man of the centre for the tail which, in the New World monkeys, plays the part of a hand."

CENTRES FOR THE UPPER LIMB, WITH THE MOVEMENTS TO WHICH THEY ARE RELATED.

4, Upper part of the ascending frontal. Movements of adduction and retraction of the arm.

5, Posterior extremity of the superior frontal convolution. Forward extension of the arm and hand.

6, Upper part of ascending frontal convolution. Supination and flexion of the forearm.

a, b, c, d, Ascending parietal convolution. Different movements of the wrist and fingers. (With these latter, retraction of the angle of the mouth is apt to be associated, owing to the proximity of the facial and oral centres.)

FACIAL AND ORAL CENTRES, WITH MOVEMENTS RESULTING FROM THEIR STIMULATION.

7, Median portion of ascending frontal convolution. Retraction and elevation of the opposite angle of the mouth.

8, Lower part of ascending frontal convolution. Elevation of *ala nasi* and upper lip, with depression of lower lip.

9 and 10, Inferior extremity of ascending frontal and posterior extremity of the third frontal convolution (Brocca's convolution). Opening of the mouth with (9) protrusion and (10) retraction of the tongue. Region of Aphasia. Action bilateral.

11, Retraction of the opposite angle of the mouth; and the head turned slightly to one side.

12, Posterior portions of superior and middle frontal convolutions. Eyes opened widely; pupils dilated; head and eyes turned towards opposite side.

13 and 13′, Supra-marginal lobule and angular gyrus. Eyes moved towards opposite side, with an upward (13) or a downward (13′) deviation. The pupils generally contracted. (Centre of vision—part of.)

14, Superior temporo-sphenoidal convolution. Pricking of opposite ear; head and eyes turned to opposite side; pupils largely dilated. (Centre of hearing.)

These are the regions which were defined by the experiments of Hitzig, and, more specially by those of Ferrier. Recently the experiments of Schäfer and Horsley have not only led to the confirmation of these localisations but also to the discovery of another important excito-motor region on the mesial surface of the hemisphere* in the marginal convolution,—excitation of which gives rise to contraction of the various trunk muscles (Fig. 13). It is desirable, however, more accurately to define the limits of these various centres, and to state the kind of movements which their stimulation evokes. These details will be found in the description, given by Ferrier, of Fig. 12, in which his experimental results with monkeys are transferred to corresponding regions in the human brain; as well as in the description given by Horsley and Schäfer of Fig. 13, having reference to the marginal convolution.

By a method of minimum stimulation which they describe, Horsley and Schäfer explored the mesial surface of the hemisphere, and soon found that positive results in the way of muscular contraction were only to be obtained from excitation of the part of the marginal convolution indicated in Fig. 13, that is, from a point about opposite the middle of the first frontal gyrus, backwards as far as the termination of the calloso-marginal fissure (c. m. s.).

Speaking of their results they say:—"A remarkable relation was found on the whole to hold good between the different parts of this convolution and the parts of the body thrown into movement by their excitation, to the effect, namely, that when the stimulus was applied anteriorly the resulting movements affected the upper limbs (and in one or two instances muscles of the head and neck); when applied near the middle of the excitable part of the convolution, the muscles chiefly or primarily affected were those of the trunk (erector spinæ, abdominal muscles, &c.), whilst when applied posteriorly, muscles of the lower limbs alone were called into action. Indeed, it appears probable that, if we regard only the results of minimal excitations, and especially if we take into account only those muscles which are primarily called into action, this rule will prove to obtain in a still more special manner, and that we may arrange the movements which are produced by stimulation of points which succeed one another from before back in the following order viz. 1.; Movements of the forearm. 2. Movements of the humerus and scapula. 3. Movements, chiefly rotation and flexion, of the upper part of the trunk and abdomen. 5. Movements of the pelvis. 6. Movements at the hip, 7. Movements at the knee. 8. Movements at the ankle-joint. 9. Movements of the toes."

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* This region is often spoken of by French writers as the 'paracentral lobule.'

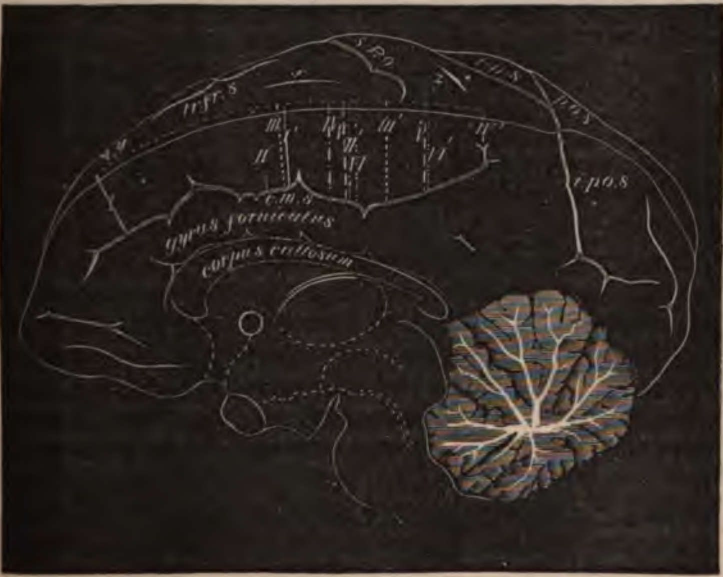

FIG. 13. DIAGRAM OF THE INNER SURFACE OF THE CEREBRAL HEMISPHERE OF A MACACQUE, SHOWING THE SITUATION OF THE SEVERAL EXCITO-MOTOR CENTRES IN THE MARGINAL CONVOLUTION [after Horsley and Schäfer].

s. Ro, Fissure of Rolando. z, Small fissure separating the upper extremity of the ascending parietal convolution from the parietal lobule. y, Small fissure near the middle of the first frontal convolution.

I-IV′, Anterior and posterior boundaries of the excitable area in the Marginal Convolution.

From what has been said above, it will be seen that whilst the characteristic movements evoked by excitation of the marginal convolution are those produced in trunk muscles as well as in muscles which connect the upper and lower extremities with the trunk, there is also obtained by its excitation movements of the forearm and of the hand and leg very similar to those which Ferrier has shown may be produced by excitation of portions of the external surface of the hemisphere. . . . It is to be noted, however, that the excitable areas for the arm as well as those for the leg occupy corresponding contiguous situations on the external and mesial surfaces of the hemisphere.

For further details reference must be made to the Preliminary Communication of Horsley and Schäfer ('Proceedings of Royal Society,' March 30, 1884).

We must turn now to a description of the different degrees of paralysis met with as a consequence of disease in one cerebral hemisphere. As before stated the paralysis is of the hemiplegic type, but in different cases it varies much in its degree of completeness according to the varying magnitude and situation of the lesion. This is found to be the case whether we have to do with cortical lesions in the excito-motor area, or with lesions deeper down, within the substance of the hemisphere—that is, in the fronto-parietal region of the centrum ovale.

In the case of lesions occurring in the excito-motor area itself it will, of course, be easily understood that a very small lesion limited to the facial centre may produce mere paralysis of the opposite side of the face, or, should the lesion be on the left side, aphasia together with paralysis of the opposite side of the face. Again, a lesion limited to the contiguous centres for the muscles of the upper limb, may produce the rare condition known as brachial monoplegia; or should the lesion involve simultaneously the centres for the upper limb as well as those for the face (Fig. 12), then we should have the not unfrequent combination of paralysis of the arm and of the face on the opposite side.

It is also obvious that a small cortical lesion may, on certain occasions, involve only the centres for the lower extremity, and we should then have another rare result in the form of a so-called crural monoplegia on the opposite side. Or a cortical lesion may involve simultaneously the centres for the upper and the lower extremities, so as to produce paralysis of these limbs on the opposite side without implication of the face.

In other cases a cortical lesion may involve simultaneously the centres for the face as well as the upper and lower limbs, and so give rise to what is known as a complete hemiplegia on the opposite side of the body.

But what, it may be asked, will be the result when the centres for the trunk muscles in the marginal convolution are involved, either alone or in concert with the other excito-motor centres just referred to? This subject will be more fully considered a little further on (p. 173). For the present I will merely say there is reason to believe that a lesion in this situation would give rise only to some amount of temporary weakness of the trunk muscles (lasting perhaps, at most, not more than two or three days), but to no definite paralysis. There are no cases on record, at all comparable to the brachial and crural monoplegias, in which the trunk muscles of one side have been paralysed as a result of a lesion in and confined to the opposite marginal convolution.

It will be easily understood that lesions in the centrum ovale, or white substance of the hemisphere, occurring not far below the several centres for the face and the upper and lower limbs, would be capable of producing just the same combinations of paralysis as have been above enumerated as consequences of cortical lesions. As a matter of fact, however, lesions are comparatively rare in these situations which do not involve the grey matter also—this being a consequence attributable in the main to peculiarities in the vascular supply of such regions.

Lower down, as the efferent fibres from the several excito-motor centres come closer and closer together, and when they pass into and constitute a large portion of the so-called 'internal capsule,' they are much more apt to be affected together (rather than separately), and thus to produce typical hemiplegic conditions.

The same may be said in regard to lesions occupying either cerebral peduncle or one half of the pons Varolii; only, as before remarked, lesions in the latter situation are very prone to extend across the middle line, and then we get a paralysis more or less completely bilateral in type.

Thus, in general terms, it may be said that a paralysis of cerebral origin is much more apt to be partial and simple when the lesion which causes it is in the cortex; and it is most apt to be extensive and compound when due to disease in the pons Varolii.

(*a*.) *Modes of Production of Paralysis in different Cases of Organic Disease of the Brain.*—Another point requires to be noticed here. The different kinds of paralysis to which we have been referring are brought about in very different ways. (1) They may be produced by a hæmorrhage which either disorganises the centres in the cortex or tears across the fibres emanating from such centres in some part of their course. (2) A hæmorrhage such as we have referred to above may cause structural damage to some cortical centres or to some part of their efferent fibres, whilst other efferent fibres may be pressed upon by the extravasation, and thus rendered for a time incapable of transmitting stimuli: it has been long recognized that recovery of functional activity may gradually take place in fibres that have been merely pressed upon, as the blood clot becomes absorbed and pressure upon surrounding parts is consequently diminished at the seat of lesion. (3) Occlusion of vessels or of some large arterial trunk, by thrombosis or embolism, cuts off at first from its proper blood supply a tract of brain tissue nearly equal to that of the area of distribution of the vessel; and if such ischæmia occurs in a motor tract its deprivation of blood will necessarily cause a cessation of function in such parts, and the establishment of a corresponding motor paralysis; soon, however, some amount of collateral circulation is set up, before the brain tissue becomes irretrievably damaged, the area of functional inertia is diminished, and a corresponding diminution takes place in the amount of the paralysis. (4) The growth of some abscess, tumour, or other adventitious product may, in other cases, be the means of destroying or compressing either centres or outgoing fibres in some part of their course—and here the signs are liable to go on increasing, though it may be slowly and fitfully. (5) Whether we have to do with hæmorrhage, softening, or with a new growth or other adventitious product, there is, in all severe cases, a more or less general increase of cerebral pressure, which is apt to cause symptoms dependent upon interference with the functions of parts more or less remote from the actual seat of lesion; if from any cause the general pressure diminishes, there will be a proportionate diminution in all such symptoms of an 'indirect' order. (6) Similarly, the disturbance of the proper balance of nerve functions, especially by lesions occurring more or less abruptly, may produce temporary symptoms, dependent upon the functional disturbance of parts not themselves primarily affected; or such 'indirect' symptoms may have other obscure relations with the primary structural damage and its immediate consequences.

One important conclusion that should be drawn from what has been said is, that only some of the more important features or symptoms presented by a patient are to be explained as direct consequences of the lesion; other symptoms will belong to one or other section of the 'indirect' category, and will be produced and, perhaps, disappear, in manners so various as to defy anything like a satisfactory explanation in detail

(*b.*) *The kind of Facial Paralysis which occurs in Hemiplegia.*—It is important to say a few words on this subject because the amount of facial paralysis existing in different cases of hemiplegia varies greatly—and for the most part in accordance with the situation of the lesion.

In some instances of hemiplegia, facial paralysis may be practically absent; this is most likely to occur in some cases of cortical disease. Still, in other cases of cortical disease, where the facial centres are themselves actually implicated, the facial paralysis is apt to be extremely well marked and persistent. Thus, it will be seen that in the case of cortical lesions, there is the greatest latitude as regards the amount of facial paralysis—though, even when it is well marked from disease in this situation it is always of the typical kind described in the next paragraph.

The typical facial paralysis that goes with hemiplegia, may be considered to be that which is commonly associated with cerebral hæmorrhage due to rupture of the principal lenticulo-striate artery (the site of the hæmorrhage being within or just outside the corpus striatum), simply because this is about the commonest cause of hemiplegia. The facial paralysis met with in these cases is usually both partial and incomplete—that is, it only affects, to an appreciable extent, some of the muscles supplied by the seventh nerve; and those which are involved, are weakened rather than wholly paralysed. The muscles chiefly affected are the buccinator and some of the straight muscles going to the angle of the mouth and lip on the paralysed side of the body; and, in consequence of the weakening of these muscles, the cheek on the affected side looks flat, the corresponding naso-labial fold is less marked, the upper lip is less arched, and the angle of the mouth on the same side is decidedly lower than its fellow. As patients and their friends often say, the face is 'drawn,' though by this expression they refer more especially to the opposite or non-paralysed side, where, owing to the unresisted action of the muscles on this side, the angle of the mouth is higher than it is on the paralysed side. This difference in level of the angles of the mouth, becomes much more marked when the patient laughs or speaks, or when, in response to our bidding, he attempts to show his upper teeth. You may note, however, that he can frown as usual, can lift his eyebrow and eyelid, and can close his eye on the paralysed side almost as well as on the other side of the face. It is only rarely that a trifling weakness of the orbicularis palpebrarum is apparent for the first day or two after the attack.

When the tongue is affected it deviates, on protrusion, slightly to the paralysed side, owing to the more powerful action of the protruding muscles on the non-paralysed side.

It is important to know these characteristics of the most common form of facial paralysis associated with brain disease because, in certain cases, as a result of such disease, paralysis of the face alone may occur without implication of either limb. Thus, such cases as these may be met with :—a patient feels giddy, finds himself either unable to speak at all for a few minutes or else only able to articulate badly ; at the same time his face may be drawn to one side though no weakness in the arm or the leg may be experienced. Such a condition may be the forerunner of a more complete hemiplegic attack, or it may have been produced by a small, non-progressive lesion. In the latter case, after two or three days all difficulty in articulating may have disappeared, the limbs may still show no sign of weakness, though the angle of the mouth may remain distinctly lower on the affected side, and the tongue may perhaps deviate slightly.

When called upon to form an opinion in regard to such a case as this it must not be forgotten that just such an unsymmetrical condition about the mouth exists in some persons more or less from birth, whilst in others it may be due to a comparative absence of teeth on one side of the upper jaw.

At other times, the face is found to be paralysed in the manner above described in concert with the arm on the corresponding side, the leg remaining unaffected. Such cases are, in the majority of instances, due to cortical or subcortical lesions, though they may also be caused by lesions lower down, perhaps in the internal capsule itself, which destroy or press upon the efferent fibres proceeding from the centres for the face and upper limb alone.

(c.) *The Distribution of Paralysis in a typical case of Hemiplegia.*— In the next group, we have to do with the ordinary typical hemiplegic cases (such as are so frequently produced by disease in or just outside the corpus striatum) in which there is paralysis of the face such as we have above described, together with a paralysis more or less complete of the arm and of the leg on the same side—but there is in these cases no marked paralysis of the trunk muscles. Why is this? and what other muscles are exempt from paralysis in such cases? These are questions of considerable interest, on which we must now say a few words.

The paralysis, as above stated, is limited to the arm, the leg, and a part of the face. It does not involve the muscles of the eyes, of the neck, the back, the thorax, and the abdomen. In explanation of this exemption Dr. Broadbent has put forward an hypothesis, which, whilst it goes far to explain the facts is also based upon sound physiological principles.

Broadbent maintains that the key to the apparent anomaly, is to be found by a comparison of the muscles paralysed with those which escape paralysis. They are then seen to fall naturally into two distinct classes. The arm is completely paralysed, and this is a part whose movements are always independent of those of its fellow. The leg, which is often less paralysed, can also be moved quite independently of its fellow—though the two limbs so very frequently move in concert. But the muscles of the eyes, neck, and trunk—those which escape paralysis—act in pairs and are almost always bilaterally combined in one or other mode. Corresponding or related muscles on the two sides of the body are, in fact, here simultaneously called into play. Thus, as Broadbent says :—" We move one arm or one leg whilst the other is quiet or executing a totally different action. We find it impossible to expand one side of the chest without the other, or to move one eye without the other, and extremely difficult to throw into action the muscles on one side of the abdomen without the other —impossible, indeed, to do this forcibly."

Now it is extremely probable that when muscles on opposite sides of the body habitually act together, and rarely or never independently, the spinal nuclei of their nerves become connected by commissural fibres. Broadbent's explanation, therefore, of the exemption of certain muscles from paralysis in cases of hemiplegia, is based upon the supposition that the two nerve nuclei in opposite sides of the cord, in relation with such pairs of bilaterally acting muscles, are so intimately connected by means of commissural fibres as to be in effect a single nucleus. He supposes that a combined nucleus of this sort will be in connection with a set of fibres from each hemisphere, and will usually be called into action by both, " but it will be capable of being excited by either singly, more or less completely, according as the commissural connexion between the two halves is more or less perfect."

This same hypothesis affords an explanation of some of the phenomena known to occur in hemichorea, as well as in cases of unilateral convulsion due to some irritation in the excito-motor region of the opposite cerebral hemisphere. Here we have to do with choreic movements or spasms in just the same limb muscles as are paralysed in a case of hemiplegia, but the bilaterally acting muscles are, where the irritation is severe enough, generally affected also, though on both sides of the body. This is precisely what might have been expected in accordance with Broadbent's theory; for, just as the cutting off of incitations from one side of an intimately connected double nucleus may entail no paralysis, because a sufficient stimulus may pass through the two halves of the nucleus from the fibres coming from the undamaged hemisphere, so may an undue amount of stimulus passing through the fibres of one side lead to spasms of the related muscles on both sides of the body.

Experimental observation has also shown that irritation of certain parts of the cortex of the brain, and more especially of the facial centres, has given rise to double associated movements on the two sides of the face. Some have imagined that this affords evidence of a double representation in the cortex of such bilaterally acting muscles, that is, that a double set of fibres pass downwards from each hemisphere of the brain; one set to excite the muscles on the opposite side, and the other the related muscles of the same side. But no independent evidence supporting such a view exists; and the facts mentioned, are capable of receiving a ready explanation in accordance with Broadbent's hypothesis—viz., in the same way that we explain the bilateral excitation of associated muscles in cases of otherwise unilateral convulsion. Any double representation in the cortex would, in short, involve a double sensory as well as a double motor representation, if the view which we have put forward in this work concerning the initiation of movements has any truth in it; indeed this hypothesis as to the existence of a 'double representation of muscles in the cortex' seems to me much less probable than the hypothesis of Broadbent.

An ingenious suggestion has been made by V. Horsley in reference to the cause of exemption of the trunk muscles from paralysis in cases of hemiplegia, based upon the results of his own and Schäfer's experiments, by which they have been enabled to determine that the marginal convolution contains the excito-motor centres for the trunk muscles. He refers especially to the common form of cerebral hæmorrhage resulting from rupture of the principal lenticulo-striate artery which, as described by Duret, courses round the under surface of the lenticular nucleus at the junction of its anterior and middle third before it passes through it and the internal capsule to gain the caudate nucleus (Fig. 14). In reference to this particular, though extremely common, site for cerebral hæmorrhage, Horsley says ('Lancet,' July 5, 1884, p. 9):—"From this position it is obvious that hæmorrhage from it will destroy the pyramidal fibres in the order of face, upper limb, lower limb, and trunk, as the focus of hæmorrhage will be below and in front of the bundles of fibres. . . . . It is obvious that the trunk fibres are as a rule only pressed upon, since if the hæmorrhage extended up as high as to completely divide them, it would be within a very few millimetres of the lateral ventricle, rupture into which would preclude further progress of the case." This is an ingenious suggestion, but it is obviously one applicable only to a certain (though very common) class of cases, and not to hemiplegia in general, from unilateral lesions in various parts of the brain. It may be true that from the mere anatomical disposition of the fibres coming from the marginal convolution, the trunk muscles are least liable to be implicated in hemiplegias due to this particular cause; but there seems to be evidence enough to show that where the motor tract of one hemisphere is completely torn across there is still nothing more than a temporary weakening of the trunk muscles

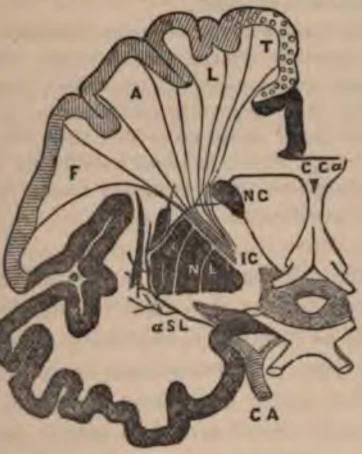

FIG. 14. SECTION THROUGH LEFT CEREBRAL HEMISPHERE NEARLY IN THE DIRECTION OF THE FISSURE OF ROLANDO, SHOWING ASSUMED RELATIVE POSITION OF FIBRES PROCEEDING FROM THE SEVERAL EXCITO-MOTOR CENTRES [after Horsley].

F, facial centres and outgoing fibres. A, centres for upper extremity with outgoing fibres. L, centres for lower extremity with outgoing fibres. T, centres for trunk muscles with outgoing fibres. CCa, corpus callosum. NC, caudate nucleus. NL, lenticular nucleus. IC, internal capsule.

CA, internal carotid artery. aSL, lenticulo-striate artery.

(Hæmorrhage occurs most frequently either in the outer part of the lenticular nucleus or just outside it.)

on the paralysed side, and that this is the rule in hemiplegia generally—it is because of its bearing upon all such cases, therefore, as well as for the explanation which it affords of the distribution of spasms in hemichorea and in unilateral convulsions (where facial and trunk muscles are so apt to be affected bilaterally) that we must still rely upon Broadbent's hypothesis as affording the best and most generally applicable explanation.

Horsley's suggestion renders no sort of explanation of these latter facts. He might say, it is not necessary that it should do so. That the excitation of cortical so-called motor centres on one side might lead, by means of commissural fibres, to the excitation of corresponding motor centres in the opposite hemisphere. But there are grave objections to this view, when we remember that according to Franck and Pitres, bilateral convulsions may still occur from irritation of one hemisphere, even when the 'motor centres' of the other hemisphere have been extirpated.

There is, however, one exception of a remarkable kind to the otherwise apparently general law, that bilateral muscles which habitually act together may be effectively stimulated through either cerebral hemisphere acting alone. No muscles are more eminently bilateral and simultaneous in their action than those concerned in the motor acts needful for the production of articulate speech, and yet, as we have previously stated (p. 118), these groups of muscles seem, as a rule, only to receive their stimulation from one cerebral hemisphere, that hemisphere being almost invariably, in right-handed persons, the left. This peculiarity appears to afford the real reason why Aphasia is generally caused by a lesion in the third frontal convolution and some other sites in the left hemisphere, and not by similar lesions in the right hemisphere.

Between the comparatively rare and imperfect form of hemiplegia in which the leg escapes altogether, and the ordinary forms of hemiplegia in which it is at first completely paralysed, all gradations are encountered in practice. But, still more rarely, we meet with an irregular form of the affection, in which, from the beginning, the leg is more decidedly affected than the arm. We are able to recognize this anomaly at once, should the paralysis of the upper extremity be from the first incomplete; though in cases of complete paralysis of the hemiplegic type, the existence of the disease in this irregular form cannot, of course, reveal itself until the patient begins to recover power. Then it is that we find the arm recovering strength more rapidly than the leg—so that, after a time, the patient may have completely regained his strength in the upper extremity, whilst the leg, instead of improving, has only become more stiff and powerless. Trousseau passed a very gloomy verdict on such cases, which certainly does not seem to be merited in all of them.

More information is wanted concerning these cases, though probably they may be referred to three categories :—

(1). We may have cases of this kind caused by lesions wholly or in part cortical, in which the cortical leg centres are proportionately much affected. Or lesions may implicate the upper part of the internal capsule in an unusual way, so as to involve the fibres emanating from the centres for the lower extremity more than those proceeding from the centres for the upper extremity.

(2). Other cases of this variety of hemiplegia are certainly met with where the lesion occurs in the pons Varolii. Of this I have seen some well marked instances, and can refer to cases verified by post-mortem examinations. The possibility of such an occurrence from unilateral lesions in this locality, is rendered more explicable by reason of the topographical separateness of the motor fibres pertaining to the arm and leg respectively in this region, as Brown-Séquard long ago ascertained.

(3). Lastly, I believe there is another class of cases in which the leg is not originally more paralysed than the arm—in which, in fact, recovery begins to take place in the ordinary way—though it may soon be arrested so far as the leg is concerned. Such cases, therefore, only seemingly belong to the variety of hemiplegia of which we are now speaking, when they are met with at a period remote from their origin; they are, I suspect, not so much due to any peculiarity of the brain lesion, as to certain quasi-accidental extensions of sclerosis from the tract of 'secondary degeneration' in the lumbar region of the spinal cord.

In some of the cases in which the arm regains power before the leg, I have found that the several segments of the limb recovered in an order the reverse of that which is usual—namely, that power was first regained over the movements of the fingers and wrists, then over those of the elbow, and lastly over those of the shoulder joint. Hughlings Jackson has expressed the opinion that this inversed order of recovery of power in the different segments of the upper extremity obtains in all, or in almost all, the cases in which the arm recovers before the leg. This is also a matter that requires further investigation. My own experience does not enable me to share this opinion. Moreover, in cases where there is no reversed order of recovery as regards the leg and the arm, I have sometimes found the several parts of the arm recover in the inverse order already mentioned, without being able at all to account for this departure from the general rule.

In lesions of the **crus cerebri** an ordinary hemiplegic condition of the opposite side of the body, including the face, is often associated with paralysis of the third nerve on the same side as the lesion. This characteristic association is most apt to be met with when the lesion affects the part of the crus contiguous to the pons.

As a rule, in lesions of the internal capsule or of any part of the hemisphere above this level (with the possible exception of lesions in the cortical facial centre itself), we meet only with that partial and temporary paralysis of the facial and hypoglossal nerves which has been already described. But as the cerebral lesion causing hemiplegia comes into close relations with, or actually invades, the pons Varolii, so is the paralysis of the muscles supplied by these nerves apt to be both more complete and more lasting. The tongue, instead of merely deviating slightly to the paralysed side, is in these latter cases, not unfrequently so much affected that, for a time, it cannot be protruded at all; there may also be difficulty of deglutition, together with a more or less copious flow of saliva from the paralysed side of the mouth. The paralysis of the face, too, is much more marked; its angle on the affected side is notably lower, and the buccinator is quite powerless, so that food tends to collect between the teeth and the paralysed cheek. There is also some obvious weakening of the orbicularis palpebrarum, so that the corresponding eye can only be partially closed. In conjunction with such phenomena as these—that is, with the set taken as a whole—there almost always exists a marked difficulty in articulation (aphemia).

This very striking group of signs may present itself (a) with the paralysis of the face on the same side as the limb palsy, though at other times it occurs (b) with an implication of the face on the opposite side of the body ('cross paralysis' or 'alternate hemiplegia'). In the latter class of cases, the paralysis of the face is sometimes almost as marked and complete as where peripheral disease of the facial nerve exists (Pt. III, E). In the former class, the paralysis may be extremely well marked in the lower half of the face, but more or less absent in the occipito-frontalis, the corrugator supercilii, and the orbicularis palpebrarum.

'Cross paralysis' or 'alternate hemiplegia' is now generally recognized as being indicative of a lesion in the pons Varolii. But this is not enough; it is indicative of a lesion in the lower or posterior part of one lateral half of the pons, because in this place a single lesion may implicate the out-going fibres of the facial (that is its root fibres) *after* their decussation in the pons, and also the channels for the conveyance of motor stimuli to the limbs *before* their decussation in the medulla (Fig. 15). A single lesion thus situated, therefore, suffices to paralyse the facial muscles on the same side and the limbs on the opposite side of the body.

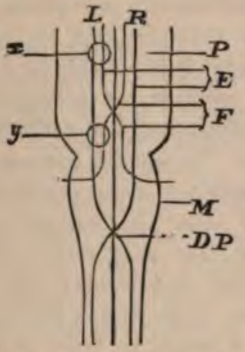

FIG. 15. DIAGRAM ILLUSTRATING THE DECUSSATION OF THE CEREBRAL FIBRES FOR THE FACIAL NERVE IN THE PONS, AND OF FIBRES FOR THE LIMBS IN THE MEDULLA.

P, pons. Mo, medulla oblongata.
DP, decussation of the pyramids.
F, fibres destined for the facial nerves.
E, fibres destined for the nerves going to extremities.
x, lesion in the upper part of the pons.
y, lesion in the lower part of the pons (which would be productive of a 'cross paralysis').

Should the lesion occur in the upper or anterior part of one lateral half of the pons, the fibres for the facial and the motor channels for the limbs are alike implicated above their respective sites of decussation, and consequently the facial paralysis, though still very marked, occurs on that side of the body on which the limbs are paralysed.

Thus, whilst 'cross paralysis' is indicative of a lesion in the pons Varolii, it by no means follows that the lesion is not situated in this region simply because we have to do with a paralysis of the face and of the limbs on the same side of the body. This is a fact which is not always borne in mind.

Again, it should also be remembered that a 'cross paralysis' may, at times, be simulated when it does not really exist. In cases of hemiplegia of the ordinary type, for instance, it occasionally happens after it has existed for a certain time that a condition of contracture is set up in the paralysed muscles. The labial commissure on the affected side then becomes raised instead of lowered, and the naso-labial furrow instead of being less than natural becomes deepened—thus the appearance of a paralysis on the opposite side is suggested and consequently the patient might seem to be the subject of a 'cross paralysis.' In such a case, however, it would be found, on close observation, that the power of movement was decidedly greater on the apparently paralysed side, and scarcely perceptible on the opposite side (or side of contracture).

According to Nothnagel, it is possible for a small lesion to exist in the pons which shall paralyse the arm completely, render the leg paretic, and yet not produce paralysis of the face on either side. He has seen two such cases. In each the lesion was situated in the upper part of the pons, very near to the crus cerebri, and close to the middle line. From these and other observations, it appears that paralysis of the extremities may occur without paralysis of muscles supplied by the facial or any other of the cranial nerves, even in the case of lesions of the pons Varolii.

On the other hand, it should be borne in mind that in some cases of lesion in the pons, paralysis of the sixth nerve (supplying the external rectus), may coexist on the same side with paralysis of the facial. In others still, there may be the coexistence of paralysis of the fifth nerve with paralysis of the facial on the side of lesion.

In another set of cases of lesion in the pons, we find paralysis of a more or less distinctly bilateral (as distinguished from the alternate) type.

In some very rare cases, double facial paralysis has been met with, associated with paralysis of the limbs on one side only. This combination is said to have occurred as a consequence of lesions involving one half of the pons only. Theoretically it would seem that such a combination of symptoms might be produced by a unilateral lesion, provided only that it extended up to, or even a trifle beyond, the middle line of the pons just in that region where the intra-cerebral fibres of the facial decussate with one another (see Fig. 15, F).

As a much more common occurrence, however, we find a more or less complete bilateral paralysis resulting from lesions in the central parts of the pons, or, in fact, from any lesions which involve both sides of this important region. A large proportion of these cases, when due to hæmorrhage or thrombosis, do not survive the apoplectic stage. But when a central lesion in the pons is slighter in extent, the patient after a time recovers from the first shock of the injury, and consciousness is gradually regained. We may then find a generalised paralysis more or less equally distributed over the two sides of the body ; sensibility may also be very notably diminished or perverted in one or more of the limbs. With this condition there is a well marked paralysis about the face, more or less marked on both sides ; distinct paralysis of the tongue, together with well marked difficulty in articulation and difficulty in deglutition.

XV. DIMINUTION OR EXALTATION OF REFLEXES.*

It is true that the superficial reflexes are commonly at first diminished in hemiplegia on the paralysed side, but we know nothing now in regard to the regional value of this sign. It may hereafter be discovered that this diminution exists especially where the lesion is situated in certain regions of the brain; and, on the contrary, does not occur when the lesion is in this or that particular region. At later periods, however, in a hemiplegic attack (that is several weeks from the date of onset), where there are signs of commencing rigidity of the paralysed limbs or where there is great exaggeration of their deep reflexes, the skin reflexes may be found to be slightly more instead of less marked than they are in the normal condition.

It is equally true that the so-called deep reflexes are commonly exalted on the paralysed side in cases of hemiplegia—when the patient has emerged from the apoplectic condition (where such a state has existed), or else at a period not very remote from the onset of the attack. The knee-jerk is distinctly exaggerated in very many cases of hemiplegia; and in a smaller number of cases ankle and even knee clonus is easily obtainable. The 'deep reflexes' are also often exalted, in the upper extremity on the paralysed side, so that a wrist-jerk may be obtained by striking the tendons on its dorsal aspect, an elbow-jerk by striking the tendons of the biceps or the triceps, or even a wrist-clonus by suddenly pressing upwards the fingers of a dependant hand.

These signs are of comparatively little value from the point of view of regional diagnosis. We know in the first place that such an exaltation of the 'deep reflexes' may occur from the presence of lesions in or pressure upon any part of the pyramidal system of fibres—from the cerebral cortex in the Rolandic region, downwards through the internal capsule, the crus, the pons, the medulla, and the opposite lateral column of the spinal cord. A lesion in any one of these regions, has no more tendency to produce an exaggeration of the 'deep reflexes' of the lower extremity, than an equivalent lesion in any other of them.

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* Concerning the nature and mode of eliciting these Reflexes see Part IV, section on 'Spinal Reflexes.'

There can be little doubt that lesions in the brain implicating some portion of the tract indicated, either directly or indirectly, are by far the most prone to lead to exaggeration of the 'deep reflexes' in a marked manner. With paralysis caused by lesions outside these regions the phenomena become decidedly less marked. We may, for instance, in a large number of cases still find an exaggeration of the knee-jerk, but be unable to elicit ankle clonus.

It occasionally happens that ankle clonus may be met with on both sides—and almost as markedly on the non-paralysed as on the paralysed side of the body—where, apparently, we have only to do with a unilateral lesion. In certain very exceptional cases it has been even more marked on the non-paralysed than on the paralysed side.

Further knowledge may doubtless, after a time, give greater precision to the indications afforded by these various conditions in favour of a lesion in this or that region of the brain; but at present we are only entitled to assume, from the existence of well marked exaltation of the 'deep reflexes,' that some conditions exist leading to a lowering of functional activity in the motor tracts in some part of the brain on the side opposite to the paralysis, or else in some part of the lateral column of the spinal cord on the same side as that on which the paralysis or paresis exists.

Exaggeration of the 'deep reflexes' may occur as a temporary condition, lasting perhaps for a few hours or days only, in association with the paresis following upon an attack of unilateral convulsions of the Jacksonian type (p. 87).

XVI. ASSOCIATED MOVEMENTS.

The occurrence of associated reflex movements (or Synkineses, as they have been termed by Vulpian) has been known for a very long period, though our information in regard to them has been notably increased within recent years.

It may be safely said that, as a rule, they are most prone to occur in cases in which the 'deep reflexes' are exaggerated and in which there exists a slight amount of contracture. Hitzig has, indeed, gone so far as to endeavour to explain the contracture itself of hemiplegic limbs, as an example of a persistent reflex contraction of this type; but this view is not commonly accepted.

The phenomena that have been spoken of under the present heading, are divisible into two distinct though related categories; we may have (*a*) reflex movements excited in the paralysed limbs or side of the face in association with movements of other parts of the body, or (*b*) associated movements occurring on the non-paralysed side of the body when active or passive movements are made in partially paralysed limbs.

(*a*).—In certain cases of hemiplegia where the lower part of one half of the face is completely unresponsive to volitional incitations, it may nevertheless be caused to contract quite as freely as the other side under the influence of a powerful emotional stimulus—one which excites to laughter, for instance. It is well known that in the majority of cases no such phenomenon occurs, and that the paralysis is, as a rule, rendered only the more obvious when the patient laughs.

Again, it has long been known that yawning, sneezing, coughing, may, in some hemiplegics, lead to more or less marked contractions in the paralysed upper extremity alone, or in that as well as in the lower extremity.

Other contractions of this type are more rarely met with. Thus, Onimus noted first that, in certain cases in which some amount of late rigidity exists, forcible closure of the hand on the non-paralysed side, or the movement of one or other of the fingers of this hand, leads to the production of exactly similar movements in the hand or fingers of the paralysed side. Westphal says that associated movements of this type are most prone to occur in cases of hemiplegia which date from infancy. These special phenomena are, doubtless, related to other facts of a more general order which may be observed very frequently, viz., that in patients in whom a slight amount of contracture exists, this condition is almost always exaggerated immediately the patient begins to perform any movement with the other arm or indeed with any part of the body. The transition from sleep to waking activity often shows this well. On awaking in the morning the upper extremity of such a patient may be comparatively flaccid, but when he begins to move a certain amount of contracture with increased flexion of the hand and arm may set in. Mental excitement is capable of causing a similar increase of contraction in the muscles of such a limb.

Hitzig has called attention to another allied condition, met with principally in paralysed limbs during the stage of recovery. When, for instance, the patient attempts to flex the arm he is obliged to make a very powerful effort—he has to overcome, as it were, some resistance before the movement can be executed. Close examination seems to show that, under such circumstances, there has been too wide a diffusion of the motor impulse, so that the extensors are called into play at the same time as the flexors, and these latter can achieve their end only by first overcoming their antagonists.

(*b*).—Nothnagel calls attention to the most common form in which this second kind of associated movement presents itself. He points out that in certain cases, when a hemiplegic patient, who has ceased to be completely paralysed, extends the partially paralysed fingers slowly and with effort, those of the unaffected side perform the same movement. Brissaud has also observed a well marked case of this type, and here the associated movement in the non-paralysed arm always distinctly preceded the volitional movement in the paralysed hand.

Last year V. Horsley called my attention to a remarkable case of this kind occurring in a child, about ten years old, who was suffering from tubercular meningitis and left hemiplegia. The child was in a condition of stupor and the paralysed limbs showed some amount of rigidity. Several times, at short intervals, I saw a passive flexion of the left forearm followed, after an interval of two to four seconds, by definite movements of the right or non-paralysed arm. The movements were always from a position of extension with abduction and supination, to one of adduction, with partial flexion, and pronation. On each repetition, the movements took place slowly and in the above-mentioned order. This case is remarkable for the complexity of the associated movement which was produced, for the facility with which it was caused to reappear, at short intervals of two or three minutes, and from the fact that it was evoked by a passive rather than by a voluntary movement. This last peculiarity makes it not quite certain whether these movements should not be included rather in the rarer category of strictly reflex movements to which Nothnagel refers (Ziemssen's 'Cyclopædia,' vol. xii. p. 121). It seems also to be related to the fact cited by Déjerine, viz., that by evoking knee and ankle clonus on the paralysed side in the ordinary manner, in certain rare cases, tremulous movements of a similar kind are caused to appear in the lower extremity on the non-paralysed side. Brissaud, moreover, says that he has often seen percussion of the patellar tendon in hemiplegics produce not only a contraction on the side struck, but also a slight jerk at the knee on the healthy side. He says that a careful analysis by the graphic method of the patellar reflexes in hemiplegics, shows that those of the non-paralysed side never present the exact physiological characters pertaining to the normal state. It is well known also (as stated in the last section), that in a certain number of cases of hemiplegia ankle-clonus can be elicited on the non-paralysed as well as on the paralysed side.

Brissaud argues, therefore, that in some way an alteration from the normal condition is induced in the lumbar region of the spinal cord on the non-paralysed as well as on the paralysed side of the body; this is shown, in the early stages, by the exaltation of the 'deep reflexes;' whilst, in certain rare cases, the condition may after some years increase so as to lead to a further manifestation in the same direction, viz., the

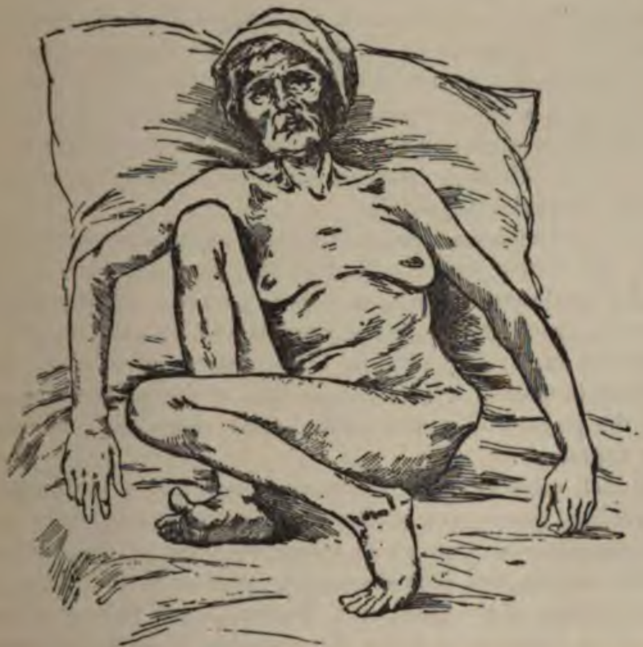

FIG 16. OLD CASE OF RIGHT HEMIPLEGIA IN WHICH A CONDITION OF CONTRACTURE HAS BECOME ESTABLISHED IN BOTH LOWER EXTREMITIES [after Brissaud].

development of contracture in the non-paralysed as well as in the paralysed lower extremity,—the patient being then, as it were, affected with a paraplegia of a spasmodic type, and thenceforth absolutely bedridden.

In 1874 a case came under my observation in which the leg on the non-paralysed side had gradually grown weaker so that a condition of partial paraplegia supervened upon a previous hemiplegic state ('Paralysis from Brain Disease,' p. 178). In this patient there was no obvious rigidity. At that time I was not aware that similar cases had been previously referred to. It appears, however, that a well marked case of this description had been commented upon by Hallopeau three years previously ('Arch. gén. de méd.,' 1871, p. 449). Charcot and Brissaud directed special attention to this sequel later on. Ormerod has lately referred to two such cases ('St Bartholomew's Hosp. Reps.,' vols. xvii. and xx.), unassociated with rigidity, and which seem to have been very similar in their character to that referred to by myself.

Brissaud says :—"There are in the wards of the Salpêtrière a great number of hemiplegic women, who have thus become paraplegic after the expiration of a certain number of years (Fig. 16). The paraplegia in such a case is complicated with contracture, and the flexion of both legs is carried to such a degree that the knees may be made to touch the chin, and extension of either of the lower extremities becomes altogether impossible."

There is one feature which is common to all the phenomena referred to in this section, and that is, that they tend to occur in hemiplegic patients in whom some amount of contracture already exists in the paralysed limbs, or else in patients in which such a condition is imminent, as is shown by the existence in them of a great exaggeration of the knee-jerk, together with the presence of a well marked ankle clonus, and, it may be, knee clonus.

In such cases we have reason to believe that a lesion exists involving some part of the cerebral pyramidal tract, and the probabilities are strongly in favour, therefore, of the existence of a more or less well-marked secondary degeneration in the opposite lateral column of the spinal cord. Beyond this, at present, we cannot go, in our attempts to define the causation of such phenomena or their association with lesions in this or that part of the brain.

XVII. RIGIDITIES OR CONTRACTURES—EARLY AND LATE.

Rigidity is met with not unfrequently in the limbs of paralysed patients. It is seen not only more frequently, but also to a more marked extent, in the arm than in the leg. This sign was much dwelt upon by the late Dr. Todd, and he distinguished two varieties of it whose pathology has been generally supposed to be altogether distinct. To the one variety he gave the name 'early rigidity,' and to the other 'late rigidity.'

In some cases hemiplegia comes and goes without the appearance of either form of limb stiffening. The muscles continue flaccid, not only at first but also whilst power is being gradually regained.

In other cases, where complete recovery does not occur, there may have been no early stiffening, although a condition of this kind is apt to manifest itself after the lapse of many weeks (late rigidity).

Early rigidity may be limited to one or two fingers, or it may be so slight as only to show itself when we proceed to move the limb. When the arm is bent, for instance, if we attempt to straighten it there is resistance on the part of the biceps ; or, if we try to flex the arm, the triceps responds, and partially resists our efforts. In other cases the rigidity soon shows itself in a very marked manner; and then the arm is strongly adducted, the elbow is in a state of semi-flexion with the forearm pronated, the wrist is slightly bent, and the fingers are flexed upon the palm. In the most marked cases any attempts to straighten the limb, or to flex the elbow more, only seem to increase the spasm ; it sets up some amount of tremor or slight tonic spasms in the limbs of one or both sides, and may cause pain as well. In less marked cases, the spasm yields considerably under steady pressure, and that, too, without producing more than a slight amount of pain.

Early rigidity is less common in the lower extremity, still it may affect the leg at the same time as the arm, and when it occurs we most frequently find the limb in a state of more or less rigid extension, the foot being in the attitude of equino-varus. It is a much less common event, in this form of rigidity, to find the thigh drawn up and the knee firmly flexed so as to bring the heel close against the buttock.

This latter mode of contraction is, in fact, decidedly rare, and commonly supervenes only at a very late period of the disease, as indicated in the last section.

Thus, when rigidity affects the upper extremities, the predominant condition is one of flexion; just as in the lower extremities the ordinary condition is one of extension.

It must not be forgotten that the same form of contracture may also implicate the lower facial muscles in a case of hemiplegia causing an appearance which at first sight, whilst the face is in a state of repose, may be suggestive of a paralysis on the opposite side of the face (p. 180). When such a patient smiles or attempts to show his teeth, however, it will be found that the lower, or apparently paralysed, side of the mouth moves most freely.

Early rigidity may show itself immediately after the commencement of a hemiplegic attack, whilst the patient is still in a state of apoplectic or epileptiform stupor. At other times, it does not present itself till two or three days after the onset of the paralysis. This kind of spasm may persist for many weeks or months, with only slight variations from day to day. The muscles do not show any signs of wasting for a long time, and they generally respond to faradisation rather more readily than those of the healthy side. We frequently find, with such a form of rigidity, that the spasm relaxes altogether during sleep, the muscles resuming their rigid condition during the act of awakening or as soon as the patient begins to move, as in other allied spasmodic states.

As to the cause of these early rigidities, the generally accepted explanation is that which was originally advanced by Todd, namely, that they are due to the 'irritative influence' of certain brain lesions or, perhaps, to a slight secondary inflammatory condition springing up in the tissues around the seat of lesion. Some such explanation may, perhaps, be the best that can be given with the view of accounting for those early rigidities which commence about the third or fourth day, and after lasting for one, two, or three weeks, again disappear. Such 'early rigidities' are, however, rare as compared with others now to be referred to.

The great majority of early rigidities are, I believe, dependent upon the locality of the lesion; and, as will be seen, they are very closely related to, if not identical with, the so-called 'late rigidities.' This holds good even for some of the cases in which rigidity shows itself from the very first, and afterwards remains, month after month, with mere slight fluctuations from time to time. Of this type I have lately seen a well marked example, where the general group of signs pointed strongly to the existence of a small hæmorrhage in the pons Varolii. This, in fact, is one of the regions in which the occurrence of lesions is apt to be associated with early rigidity. It is likewise met with where lesions exist in the thalamus, or just outside this body and the posterior extremity of the corpus striatum; and also in association with lesions in, or contiguous to, the excito-motor area of the cortex.

Early rigidity is, doubtless, in the majority of cases a grave symptom, because its occurrence and persistence for a long time renders the chance that the patient will recover power in these parts comparatively small. After a time, indeed, if the spasm does not relax, the condition of the limb differs in no way from what it presents in that form of rigidity to which we are now about to refer.

What has been termed **late rigidity** supervenes generally after many weeks, though previously all the muscles may have been in a flaccid condition. This form of rigidity, like the other, is commonly more marked in the arm than in the leg. It may be limited to the flexors of the fingers, or it may extend to other muscles, so that in the most extreme cases the arm becomes immovably bent at the elbow, the wrist is firmly flexed, and the fingers are pressed against the palm of the hand—so forcibly, it may be, as to cause some amount of ulceration in this situation. Attempts to straighten the contracted parts when in this condition, are apt to cause much pain, and if the rigidity has been of long duration are usually ineffectual. In the leg a similar contraction of the hamstring muscles may exist, with rigid tendons; and there may likewise be more or less rigidity and contraction of muscles about the ankle joint, the foot being in the attitude of equino-varus. Wherever this form of contracture exists to a well marked extent, the affected muscles ultimately, though perhaps not till some years have elapsed, become notably wasted.

In the early stages of its manifestation it should be distinctly understood that this 'late rigidity' differs in no way from the form of 'early rigidity' which we have stated above to be the most common. It begins in a gradual manner, at first presenting itself only from time to time, though ultimately it becomes more and more persistent in the parts affected. Nothing but the most artificial distinction can indeed be made between these two groups of cases, they really glide into one another almost imperceptibly—that is to say, a case which begins as one of so-called 'early rigidity,' may terminate without any break in its continuity in a condition of fairly well marked late and persistent rigidity.

Though a condition of 'late rigidity' is also apt to become established in paralysed facial muscles after the lapse of a variable time, and thus to produce sometimes a deceptive appearance of paralysis on the opposite side of the face, to which we have previously referred, it has never been known to occur in the trunk muscles.

For some time after the commencement of late rigidity, it is found to relax to a considerable extent during sleep, but to become re-established in the morning, as soon as the patient begins to move. In the course of some weeks, or it may be months, however, such relaxations at night gradually cease, and finally the limb remains in a condition of persistent rigidity—and then attempts to straighten it by even a considerable amount of force are no longer successful.

Both late and early rigidity are commonly associated with that exaltation of the 'deep reflexes' to which we referred in the last section but one. The explanation of this association will be given later on—that is, towards the end of Section XIX.—since there can be little doubt that both sets of conditions are commonly, though perhaps not invariably, connected with secondary degenerative changes set up in the lateral columns of the spinal cord, owing to the existence of disease involving higher parts of the pyramidal tracts. This is the opinion now most commonly entertained.

It was originally held by Dr. Todd that late rigidity was, in some unexplained way, brought about by means of 'irritative influences' springing up in connection with the process of cicatrisation taking place at the seat of brain lesion. He did not attempt to work out in detail how this assumed irritative process was transmitted to the muscles.

Hughlings Jackson has, however, put forward another interpretation differing notably from that which is commonly held. He believes, for instance, that late rigidity is a consequence of the cutting off of cerebral influence from the affected muscles, and of the consequent unantagonised action upon them of cerebellar centres. It will be better to defer further reference to this view, and to my own, which does not differ much from it, till we come to the close of the section dealing with 'Secondary Degenerations.'

XVIII. POST-HEMIPLEGIC CHOREA, ATHETOSIS, AND OTHER DISORDERS OF MOVEMENT.

Tremors and choreic movements have been known sometimes to precede, but more frequently they accompany or follow a hemiplegic attack. Special attention was called to this subject about ten years ago by Weir Mitchell, and shortly afterwards by Charcot.

At the commencement of his paper Weir Mitchell stated ('Amer. Jrnl. of Med. Scien.,' Oct., 1874) that the objects of his communication were to show, "That in adults who have had hemiplegia and have entirely recovered power, there is often to be found a choreal disorder, sometimes of the leg and the arm, usually of the hand alone. That it may exist in all degrees with partial loss of power, and with full normal strength. That it may consist in mere awkwardness, or exist to the degree of causing involuntary choreoid motions of the part."

A typical case of **post-hemiplegic chorea** has been reported by Charcot, to which we will now refer, as it illustrates all the phenomena that are of most importance. The patient was a woman 44 years of age, who, after an apoplectic attack, was found to be completely paralysed on the right side, the limbs being flaccid. After six months the patient began to walk a little, and also to regain power over the corresponding upper extremity, though in this limb the movements were hampered by a peculiar sort of trembling. The same kind of trembling subsequently affected the leg, but not the face. This peculiar tremor of the paralysed limbs had existed for ten years when she came under observation, and it was described in the following terms.

When the patient is tranquil and lying at rest, the movements of the affected limbs are only very slight, and not very different from those which occur in a case of ordinary chorea. The hand is agitated by slight abrupt and purposeless movements, whilst the fingers are similarly flexed and extended. The lower extremity is also the seat of obvious movements; thus, abrupt and involuntary contractions of the anterior thigh muscles cause rhythmic elevations and depressions of the patella. The foot is at the same time in constant movement from side to side, varied by flexions and extensions. When, however, the patient attempts to perform some movement, the choreiform agitation becomes extreme and closely approximates in character to that met with in disseminated sclerosis. In the act of carrying a glass of water to the mouth, for instance, the arm is agitated by very extensive rhythmic movements, which makes the attainment of the end in view very difficult.

When the patient attempts to stand or to walk slowly, supported by a stick held in the left hand, the whole body becomes agitated by movements, brought about by sudden alternate flexions and extensions of the right knee and right ankle joints. In this position, the patient's arm remains motionless so long as she keeps her hand strongly applied to her side; otherwise it would be, like the lower extremity, constantly in movement.

The more the attention is concentrated upon these movements, with a view to checking them, the worse they become.

This condition is generally associated with a more or less marked hemianæsthesia of the affected side.

In other cases it happens that the choreiform movements manifest themselves in the limbs, or the limbs and face, before actual paralysis declares itself. A patient may be seized with vertigo, together with numbness in the limbs or in the whole of one side of the body, after which choreiform movements show themselves in the affected side. This state of things may last for hours or days before the onset of paralysis, which again is apt to be associated with distinct hemianæsthesia. This is sometimes spoken of as præ-hemiplegic chorea.

The fact of the almost constant association of hemianæsthesia with these cases of pre- or post-hemiplegic chorea soon arrested attention, and led to the conjecture that the lesion in such cases would be found in the posterior part of the hinder segment of the internal capsule (which is known to be affected in hemianæsthesia), however it might also extend beyond the confines of this region. In autopsies that have since been made, this conjecture has been realized in a very complete manner. Raymond has written an important memoir on this subject. He has collected and analysed eight of these cases in which an autopsy was made; he has also attempted to study the subject experimentally by operating, after the manner of Veyssière, upon the posterior part of the internal capsule. He agrees with Charcot in the view that the seat of the lesion in pre- or post-paralytic chorea is, like that for hemianæsthesia, in the posterior part of the internal capsule, but that the lesion bears more especially upon some bundles of fibres lying just in front and to the inner side of those whose injury produces hemianæsthesia, in contact with the posterior extremity of the thalamus.

Curiously enough, this particular region has, as Grasset points out, an independent blood supply. The lenticulo-optic branch of the Sylvian supplies the posterior part of the internal capsule and the external and anterior aspect of the optic thalamus. But it is the posterior optic branch of the posterior cerebral which supplies the posterior part of the thalamus, and precisely the region assigned for lesions that produce the choreic affection to which we have just been referring. The lesions found have generally been hæmorrhages, but occasionally foci of softening have been met with involving the posterior part of the thalamus together with some of the fibres of the contiguous posterior portion of the internal capsule.

Whilst the region above named is the most common site of the lesion in post-paralytic hemichorea, it is evident from other cases which have been recorded that it may also be due to an affection of the same set of fibres lower down—that is, either in the crus cerebri, in the pons Varolii, or in the medulla. One of the first of these cases that came under my own observation, early in 1874, was that of a woman in whom there existed, almost certainly, a lesion in the crus cerebri ('Paralysis from Brain Disease,' pp. 157, 149). It is true that the movements were not quite typical in this case, since they partook rather of the nature of the rhythmic movements of the hands and toes met with in paralysis agitans. The woman became affected with tremors in the left hand and wrist when she had regained some amount of motor power. The movements consisted principally of slight, rapid and partial pronations and supinations of the hand, together with twitchings of the extensor tendons at the wrists. A few weeks later tremors commenced in the toes of the left foot, consisting principally of rapid flexions and extensions. The tremors were always decidedly worse when the patient was under observation, and entirely ceased during sleep. Grasset and Leyden have also observed somewhat similar cases.

Weir Mitchell expresses the opinion, "that the younger the person when paralysed, the more probable is the occurrence of choreal developments, so that in many cases of infantile deformity the choreal troubles remain as the chief difficulty long after there has been a restoration to full muscular power."

Finally, we may call attention to the fact that this præ- or post-paralytic hemichorea, often bears the closest resemblance to the hemichorea that occurs so commonly in children as a neurosis, unconnected as yet with any definite lesion. As we have already seen, exactly the same kind of resemblance also exists between the cerebral hemianæsthesia which is produced by definite lesions in the posterior part of the internal capsule and what is commonly known as 'hysterical hemianæsthesia,' that is, a mere functional disease or neurosis (p. 152). The fact of the independent vascular supply for each of the contiguous regions believed to be at fault in these two affections—post-hemiplegic chorea and hemianæsthesia—is a point of considerable interest well worthy of being borne in mind.

Athetosis.—There can be little room for doubt but that the peculiar condition named athetosis by Hammond in 1871 is only a special form of post-paralytic chorea. He described it as characterised by an incessant movement of the fingers and toes, and by the impossibility which the patient experiences of maintaining them in any fixed position whatsoever. The movements are absolutely irregular in kind and order, and in this respect differ altogether from the rhythmic oscillations met with in paralysis agitans. The successive movements take place slowly, but they have a rather considerable range; they are not always limited to the fingers and toes, but may extend to the hand and foot and sometimes even to the muscles of the neck and face. At other times such movements may be present only in the fingers. The movements persist during rest (sometimes they even continue during sleep), though they are aggravated by excitement, by movement, or even by directing the attention to them.

This condition usually follows hemiplegia—that is, in a very small percentage of such cases—becoming developed in one or both of the previously paralysed limbs. It has not, however, always such an origin. Occasionally it follows some mere convulsive attack without paralysis. It has even been met with by Rosenbach and Leyden in cases of locomotor ataxy; so that there is probably much truth in the view of the former observer that athetosis is a mere symptom which, like nystagmus, may present itself as one of the signs of several different diseases.

Sometimes athetosis occurs as a bilateral affection, and in this form it is most common in imbecile children. Several observers have met with this kind of association, which occasionally dates from birth. This double athetosis is, as Grasset says, best regarded as a variety of chorea, and its consideration need not further detain us. Details concerning these various forms of athetosis are to be found in an able monograph by Oulmont ('Étude clinique sur l'athétose,' 1878).

What we are here concerned with more especially is hemiathetosis, viz., that form which occurs as an accompaniment or sequence of hemiplegia, and this too is the form which is by far the most common. It has been noted in several of the cases that a more or less distinct hemianæsthesia has existed on the affected side. There have also been subjective sensations of numbness, and the affected limbs have often been colder and more livid than natural.

It may be pretty confidently affirmed that post-hemiplegic hemiathetosis, is only a variety of post-hemiplegic hemi-chorea, and further that it is dependent upon a lesion involving a closely similar brain region. Further facts, however, are needed for the positive solution of this latter question.

Hemiataxia.—It will, perhaps, be more convenient to refer now to another rare disorder of movement occasionally met with in cases of hemiplegia associated with hemianæsthesia, or even with this latter condition existing alone in a more or less complete form. By referring to it in this place, we bring together, for comparison, the different anomalies met with in the nature of post hemiplegic movements—voluntary as well as involuntary. The varieties to which we have previously referred have been in the main of the involuntary type; but that of which we are now about to speak is a disorder occurring in the execution of voluntary movements.

In the majority of cases of cerebral hemianæsthesia, whether produced by organic disease or of functional ('hysterical') origin, there may be no ataxia or incoordination of movement whatsoever. On the other hand, in certain rare cases, this condition does exist in a typical manner, as in one that was recorded many years ago by Demeaux. I have recently had under my care a case of double hemianæsthesia with paresis, in which a similar condition existed in both upper extremities. There were no rigidities, no tremors nor involuntary movements of any kind; but when, with closed eyes, the patient attempted to execute any particular movement, it was markedly incoordinate and irregular. Again, when her eyes were covered, this patient had no knowledge as to the position in which her upper extremities were left after a series of passive movements.

Gowers has recorded a case in which there had been an apoplectic attack followed by right hemiplegia, about eighteen months before the patient came under observation. There was at that time only slight paresis remaining, without rigidity and with no spontaneous movements of any kind. There was marked incoordination of the right arm during voluntary movement, which became exaggerated when the eyes were closed. Tactile sensibility was diminished in this arm though sensibility to pain was normal. In another somewhat similar case, Gowers found " a puckered cicatrix " extending through the left thalamus from one side to the other.

Grasset has recorded as a case of hemiataxia, one which probably ought not to be so included. There were no spontaneous movements, and no mention is made of the existence of any degree of hemianæsthesia. There was extremely well marked incoordination during the execution of voluntary movements; but Grasset says expressly that this incoordination was not increased when the eyes were closed. It was a case, moreover, in which three lesions existed in the opposite hemisphere. The movements met with were exactly of the same type as those encountered in disseminated sclerosis, which are indeed closely related to those of post-paralytic hemichorea. Although these latter movements go on to some extent when the patient is at rest, they are always greatly aggravated on the occurrence of voluntary movements. The fact that there was no increase in the incoordination in Grasset's patient when the eyes were closed, shows that the essential characteristic in all ataxic cases was absent.

We would suggest that these cases of hemiataxia ought really to have been considered under the head of hemianæsthesia, because the ataxia of cerebral as well as that of spinal origin is probably due to the cutting off of certain ingoing impressions (mostly of an unconscious type) derived from the muscular system, anterior to their arrival in the kinæsthetic centres of the brain (p. 108). When such kinæsthetic impressions are defective, the brain has to rely unduly on the sense of sight; hence on such a patient being told to close his eyes, the incoordination of his movements at once becomes aggravated.*

We are not at present aware of the exact course pursued in the brain by those fasiculi of fibres which conduct these unconscious impressions pertaining to the kinæsthetic sense. The fact that in many cases of hemianæsthesia this particular bundle of fibres does not appear to be involved, and that in other rare cases it is involved with the rest, would seem to show that it is somewhat separated from the great mass of the sensory fibres composing the posterior extremity of the internal capsule, and yet that it is not very far removed therefrom. The comparative isolation of the fibres conveying these impressions is further rendered probable by a consideration of some cases recorded by Landry in which an ataxic condition existed alone—that is, independently of motor or ordinary sensory paralysis.

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* We believe that **cerebellar ataxy** is brought about in a totally different manner. In it we have to do with a defect, not in the inception of movements, but at a later stage, that is, in the actuation of movements. Ataxia of cerebral or of spinal origin is primarily due to a sensory defect (if we may use such a term in regard to the want of unconscious ingoing impressions), whilst cerebellar ataxy is primarily a motor defect. In the latter case a cutting off of sight impressions does not materially aggravate the incoordination. Certainly a patient with cerebellar disease does not become unsteady when his eyes are closed, as is the case with a patient suffering from locomotor ataxy.

XIX. SECONDARY DEGENERATIONS IN THE BRAIN AND SPINAL CORD.

It has been known more especially since the researches of Türck in 1851, that cerebral lesions damaging the motor tract on one side—either in the internal capsule, the crus, or the pons Varolii—give rise to a well marked 'Secondary degeneration' in the motor tracts of the brain below the seat of lesion, and also in the opposite lateral column of the spinal cord. Many years, however, anterior to the date of Türck's researches, a case of this kind was described by Cruveilhier and illustrated in one of the plates of his 'Anatomie Pathologique' (1835-42).

The secondary degenerations which occur in the brain and spinal cord are now known to be essentially similar in their pathogenesis to processes that take place under certain conditions in the peripheral nerves. These latter changes happen to have been fully studied and explained by Dr. Augustus Waller in 1851. Some years later Schiff, and then Phillipeaux and Vulpian, pursued the same line of enquiry.

Secondary degeneration in nerves and nerve centres have however attracted much more attention during recent years, because their importance has been more fully recognized. We will in the first place say something as to the nature of the processes by which they are caused, and then enter into some details concerning their common sites or modes of distribution in the brain and spinal cord.

Waller ascertained that when a motor nerve is cut across, the part below the section (being severed from central ganglionic influences) begins to undergo a process of fatty degeneration throughout its whole length. The white substance of the nerve speedily alters in its appearance when examined by the microscope; in eight days these changes have already become marked, and they go on steadily increasing. It at first becomes more opaque and clouded, then begins to break up into large irregular fragments; the fragments becoming progressively smaller and smaller, till eventually they seem to become resolved into small globules and granules of a fatty character. This latter stage may be reached in from three to five weeks, and then we have within the sheath of each nerve mere fatty débris of a granular or molecular character surrounding the axis cylinder which still persists for a considerable period—certainly for six months or more. This process of degeneration does not advance successively from point to point, starting from the place of section towards the periphery; on the contrary, it seems to occur simultaneously throughout the whole length of each nerve fibre below or on the distal side of the point of section.

It was also ascertained that when the anterior roots of any of the spinal nerves were cut across, the portions of the roots above the section (that is, still in relation with the cord) preserved their normal appearance, whilst all the fibres of these anterior roots below the section underwent a similar simultaneous degeneration throughout their whole length. Thus beyond the spinal ganglia these degenerated efferent fibres would be found intermixed with the undegenerated ingoing fibres, on their way to the posterior roots of the several spinal nerves.

When the posterior roots of the spinal nerves were cut, the results were of a different kind. The part of the root below the section—that is, between it and the ganglion—underwent no appreciable change; nor did the sensory fibres beyond the ganglion, that is, between it and the periphery, show any change. But the part of the posterior root above the section, the proximal portion, underwent the atrophic change previously described, and on account of this its component fibres could be traced, on microscopical examination, ascending for various short distances within the posterior columns of the cord, and finally losing themselves in the grey matter.

Waller came to the conclusion that these changes, now commonly known as 'secondary degenerations,' were due to the severance of the connection between the portions of the nerve fibres cut across and their ganglionic attachments—to the interruption, in short, of some controlling nutritive influence which is usually exercised over the whole length of each nerve fibre by the nerve cell at one of its extremities, viz., that from which it proceeds. This influence is usually exercised in the direction of the physiological action of the fibre. Thus Waller concluded, from the facts above related, that the nerve cells presiding over or controlling the nutrition of the motor nerve fibres were situated in the "grey matter of the cord," whilst those for afferent fibres, on the other hand, were situated in the ganglia on the posterior roots.

Subsequent observations have shown that the course of secondary degenerations in the efferent or motor tracts of the brain and spinal cord, also invariably take place in a downward direction from the point where the destructive lesion operates, and in situations which will subsequently be indicated; also that bands of degeneration occur in an upward direction in certain regions of the spinal cord, which, as yet, have been traced but a very short distance into the brain; these latter degenerations involve fibres which, in all probability, serve for the transmission of afferent impressions of different kinds.

From what has been said it will be obvious that the so-called 'descending degenerations' in the brain and spinal cord will be analagous to the degenerations that occur in motor nerves; whilst the 'ascending degenerations' in these parts will correspond with those that occur when the posterior roots of the spinal nerves are cut across.

In the **brain**, so far as we know at present, descending degenerations occur only in the fibres which issue from the excito-motor or so-called 'Rolandic area' of the cortex. Nothing is yet known about such degenerations occurring in the course of efferent cerebellar fibres, though it is highly important that some investigations should be made in this direction.

Of ascending degenerations in the cerebrum, moreover, nothing is as yet known. Some degenerations of this type have, however, been traced for a short distance from the spinal cord into the cerebellum, by way of its lower peduncle.

In the **spinal cord** descending degenerations occur (1) in the pyramidal tracts, which are directly continuous with part of the fibres issuing from the Rolandic area of the cortex, and (2) in the short commissural fibres of the antero-lateral columns which connect certain upper with other lower parts of the central grey matter, throughout the whole length of the spinal cord.

Ascending degenerations in the spinal cord occur (1) in the fibres composing the columns of Goll, which terminate above in certain nuclei in the medulla oblongata (the trophic cells of these fibres being situated below, at different levels, in the grey matter of the cord); (2) in the direct cerebellar tracts, in which are contained the fibres above referred to as terminating in certain parts of the cerebellum; and (3) in certain short commissural fibres situated in the outer parts, or root zones, of the posterior columns, whose trophic cells seem to be situated at their lower extremities. These ascending degenerations will be more fully described elsewhere, in Part IV. of this work.

Secondary degenerations, whether they occur in the brain or in the spinal cord, have a fascicular or band-like character, such a disposition being due to the fact that these changes take place in certain cerebral or spinal tracts in which many fibres are aggregated together having functions of a like kind. If, therefore, a lesion occurs which interrupts the continuity of more or less of the fibres entering into one of these physiological bands, the same kind of degeneration begins to take place in all the injured fibres composing such an aggregate, throughout their whole length, below or above the seat of lesion, according as the particular set of fibres involved have efferent or afferent functions.

The changes that occur in these degenerating tracts are of three kinds:—(1). There are degenerative and atrophic changes in the nerve fibres themselves, substantially similar to those which occur in the peripheral nerves, leading to the destruction of the white substance around the axis cylinders. According to Bouchard the axis cylinders of the nerves also soon disappear in the tracts of secondary degeneration, since neither he nor any previous observer had been able to find them. But shortly after the publication of his admirable memoir I succeeded in finding denuded axis cylinders existing in very well marked tracts of degeneration in the spinal cord of a man who died just six months after the date of certain concussion-lesions occurring in that organ ('Med. Chir. Trans.,' 1867). (2). After the degenerative changes have advanced to a certain extent, an abundance of large granulation corpuscles begin to form between the wasting nerve fibres, so that, for a long time, no actual diminution in bulk occurs in the affected tract. (3). The next important change which occurs in the band of degeneration is one of a secondary order, and consists in an exuberant overgrowth of the neuroglia between the axis cylinders and around the granulation corpuscles (Fig. 17). This change does not show itself to any marked extent till the others are fairly well advanced, but it

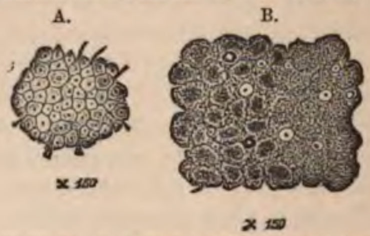

FIG. 17. SECTION THROUGH A TRACT OF SECONDARY DEGENERATION IN THE SPINAL CORD. (× 150 diameters.)

A. Section through a healthy portion of the lateral column of the cord.

B. The right half of the figure represents the appearance of a thin section through a tract of secondary degeneration, when mounted in glycerine, showing large granulation corpuscles in abundance; whilst the left half of this same portion represents the appearance of a similar section when mounted in Canada balsam, when the granulation corpuscles are rendered invisible, and the locular overgrowth of connective tissue comes out all the more distinctly. In the midst of the latter, some sections of unaltered nerve fibres are seen, and also sections of vessels with thickened walls.

is one which persists and grows more and more prominent after a year or two, when the granulation corpuscles gradually disappear, and a tract of secondary sclerosis is left as a dwarfed and changed representative of the previous band of nerve fibres. Without going into details these are the essential processes—so that we have in the tracts of degeneration, diseased and atrophied nerve fibres, thickly interspersed with large granulation corpuscles, and closely surrounded (finally strangled) by an overgrowth of connective tissue. There is nothing of the inflammatory type about the change; it is, in my opinion, essentially degenerative from beginning to end. The fact that occasionally the sclerotic process extends beyond its original limits, is not peculiar and does not in the least militate against this view, or suffice of itself to confer upon such a change an irritative or 'inflammatory' character. Yet some authorities speak of the process of secondary degeneration as a 'chronic myelitis.'*

One most interesting point about these secondary degenerations is, that for the most part they cannot be detected by any mere naked eye examination of the cord or brain at the time of the autopsy. For a long period there may be no appreciable alteration in colour or in bulk. As long as the granulation corpuscles are abundant the diseased tract may present a dead white colour scarcely if at all different from that of contiguous healthy columns; and at this stage, also, there is commonly no shrinking. Later on, the diseased tracts lose their dead white appearance, and the tissue appears of a greyish or bluish-white colour; then also some amount of diminution in bulk begins to manifest itself. In the early stages, moreover, there is no appreciable alteration in consistence in the diseased tract. Taking these facts into consideration, it is easy to understand that, at the time of the autopsy, such changes may escape detection even after a most careful scrutiny.

But in a week or two, after the parts have been immersed in a solution of chromic acid or bichromate of ammonia, the tracts of disease can be detected with the greatest ease on the surface of freshly cut segments of the cord. This is due to the fact that the fattily degenerated tracts do not become coloured by the preservative fluid; they retain their original dead white colour, whilst the adjacent healthy nerve tissue becoming stained by the preservative fluid presents the usual dark, yellowish brown hue. By this difference in colour alone, therefore, even a mere naked eye examination enables us easily to trace out the main course of these degenerations; though this may be done still more completely by examining, with a low power of the microscope and by reflected light, rather thick sections mounted in glycerine, when the aggregations of granulation corpuscles are so easily recognized that their distribution may be very efficiently traced. When thin sections are mounted in dammar or Canada balsam the granulation corpuscles are no longer visible and their place is only indicated by the loculated arrangement of the overgrown neuroglia (Fig. 17, B).

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* Charcot in one place in his 'Localisation of Cerebral and Spinal Diseases' speaks (p. 136, *note*) of the process having an irritative nature, on the ground that the sclerosis occasionally spreads from its original seat so as to involve the anterior cornu, whilst later on (p. 204) he seems to doubt the reality of any such extension. He also speaks of this process as a "chronic myelitis occurring in the lateral column in consequence of the cerebral lesion" (p. 139).

After what has been said it would be needless to insist upon the important aid which degenerations of this kind afford, in helping us to unravel some of the most difficult problems in connection with the functional anatomy of the nervous system. This method is second to none for the accuracy of its results.

Descending Secondary Degenerations.—We shall, for the present, restrict our attention to the secondary degenerations that are initiated by brain lesions, and which are apt to extend downwards from the brain into the antero-lateral columns of the spinal cord. These are all of them so-called 'descending secondary degenerations,' and their occurrence marks out for us the most important tracts along which efferent or motor impulses find their way through the cerebrum to the various muscles of the body.

The first important question which presents itself is, therefore, (*a*) as to the situations in the brain, in which the occurrence of destructive lesions is apt to be followed by these descending secondary degenerations; and the second is, (*b*) as to the principal differences in degree or extent of area involved by the secondary degenerations that are to be met with, in accordance with variations in the situation and extent of the primary lesion.

It should be premised, that hæmorrhages and softenings occurring

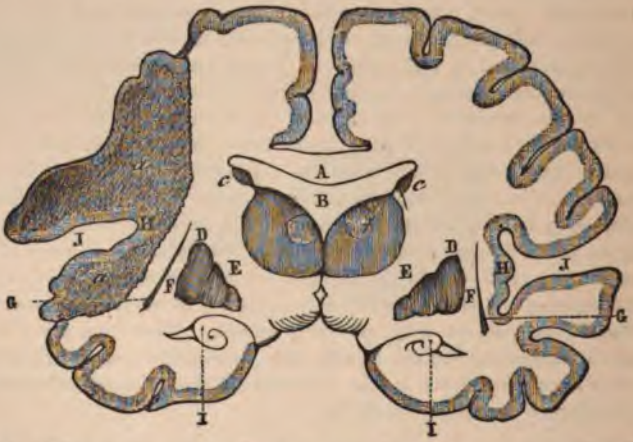

FIG. 18. A SOFTENING OF THE CORTEX OF THE LEFT HEMISPHERE WITHOUT PARTICIPATION OF THE CENTRAL MASSES, WHICH GAVE RISE TO WELL MARKED SECONDARY DEGENERATION [after Charcot].

H, circumscribed area of Softening. J, fissure of Sylvius. E, internal capsule D, lenticular nucleus. F, external capsule. G, claustrium.

in the regions of the brain about to be mentioned, are especially apt to be associated with secondary degenerations, whilst tumours are not—apparently because they often push aside and separate, instead of destroying, the continuity of the nerve fibres implicated.

Very many parts of the brain may be injured without giving rise to secondary degenerations. It has now been pretty definitely ascertained that a mere superficial lesion of the cortex (such as may occur frequently in association with meningitis) does not produce a secondary degeneration, in whatsoever part of the cortex it may occur. If, however, the lesion involves the whole thickness of the cortical grey matter, or this together with a portion of the subjacent white matter (such as may be met with so frequently in the form of a softening occasioned by occlusion of one of the vessels of the cortical system), it has been found that secondary degenerations occur only when such lesions implicate some portion of the cortex comprised within the excito-motor or so-called Rolandic area (see p. 165). The only qualification needful to be made here is, that up to the present date it has not been definitely ascertained, from observations in man, that lesions limited to the marginal convolution give rise to any such degenerations. Still this evidence may in time be forthcoming.

Lesions of the grey matter in parts of the frontal and of the parietal lobes outside the Rolandic area, as also lesions in the occipital and temporal lobes, have been shown not to be associated with secondary degenerations.

Again, it was ascertained long ago by Türck that lesions strictly limited to the central grey masses—that is, to the caudate and lenticular nuclei of the corpus striatum and to the optic thalami, do not give rise to secondary degenerations. The same observer, however, ascertained that lesions which involved the fibres of any portion of the internal capsule lying between the two nuclei of the corpus striatum are capable of giving rise to descending degenerations, whose extent depends upon the amount of destruction of the fibres of the capsule. In regard to this point our knowledge has since been advanced, and made much more precise, by Brissaud and others.

Similarly, it is now known that lesions occurring in any part of the centrum ovale which directly intervenes between the above specified Rolandic area in the cortex and the internal capsule (but especially when they occur in parts just above the internal capsule, where the fibres of the pyramidal tract become more closely aggregated), may also cause such degenerations. The more the situation of the lesion in the centrum ovale is removed from the internal capsule, that is, the nearer it is to the cortex, the larger it must be in order to produce an equivalent amount of secondary degeneration.

We must now refer to the precise situations occupied by different descending secondary degenerations in the internal capsule itself, together with the related sites in the crus cerebri occupied by the several prolongations of such areas of degeneration. Brissaud has recorded some important cases illustrating these points.

In one interesting case which came under Brissaud's observation an old focus of softening was found in the anterior half of the lenticular nucleus which also destroyed most of the anterior segmen of the internal capsule. In this case a tract of degeneration was found occupying the inner third of the foot of the peduncle, except for a small bundle of the innermost fibres which remained in their normal condition. This tract of degeneration occupying the

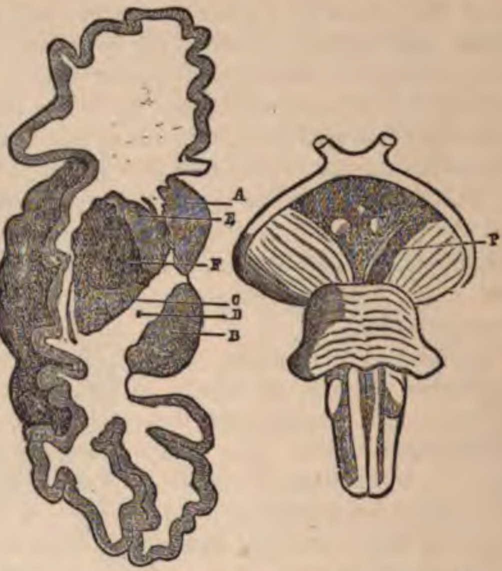

FIG. 19. DISEASE OF THE LENTICULAR NUCLEUS AND OF THE ANTERIOR SEGMENT OF THE INTERNAL CAPSULE, CAUSING SECONDARY DEGENERATION IN THE INNER THIRD OF THE FOOT OF THE CEREBRAL PEDUNCLE [after Brissaud].

A, caudate nucleus. B, optic thalamus. C, posterior and healthy part of the lenticular nucleus. D, posterior segment of the internal capsule. E, lesion of the anterior segment of the internal capsule. F, cyst presenting the form of the lenticular nucleus. P, degeneration of the internal fibres of the crusta.

anterior segment of the internal capsule could not be traced lower than the pons Varolii, and, according to Brissaud, degeneration in this region and in the internal third of the peduncle has always been associated with intellectual disorders.

The next to be referred to was one in which, with an extensive recent softening in the left hemisphere, there was an old focus of softening exactly limited to the knee (or genu) of the internal capsule. A streak of secondary degeneration was observed in this case between the internal and the middle third of the foot of the peduncle. According to Brissaud, in some long standing cases of aphasia a tract of degeneration extends downwards from the cortex which passes through this same region—that is through the knee of the internal capsule. This, therefore, may pretty safely be considered as the part of the internal capsule which is organically connected with the third frontal convolution; and we may also conclude that the fibres pass from this region through the crus cerebri in the situation above indicated.

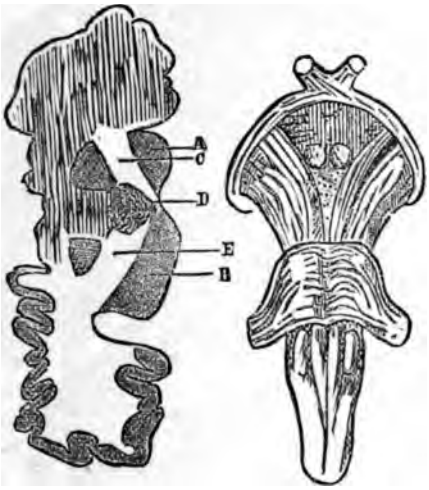

FIG. 20. RECENT SOFTENING OF THE FRONTAL LOBE, THE ISLAND OF REIL, AND THE MIDDLE THIRD OF THE LENTICULAR NUCLEUS, TOGETHER WITH AN OLD FOCUS OF SOFTENING OCCUPYING THE KNEE OF THE INTERNAL CAPSULE [after Brissaud].

D, old focus of softening occupying the knee of the internal capsule. A, caudate nucleus. B, optic thalamus. C, anterior, and E, posterior segment of the internal capsule.

From what has been said above it will be seen that the statement formerly current, to the effect that the anterior two-thirds of the internal capsule contain afferent or motor fibres, whilst the posterior third contains afferent or sensory fibres, is not quite correct. The more recent investigations of Flechsig, Charcot and Brissaud have necessitated some modification of this view.

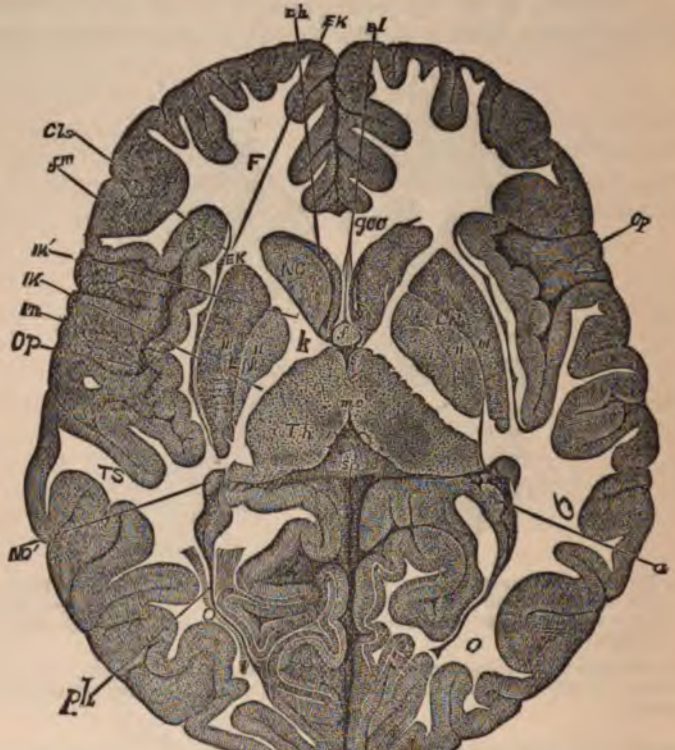

FIG. 21. HORIZONTAL SECTION OF THE BRAIN OF A CHILD NINE MONTHS OLD, THE RIGHT SIDE BEING AT A SOMEWHAT LOWER LEVEL THAN THE LEFT HALF [after Flechsig].*

F, frontal; TS, temporo-sphenoidal; and O, occipital lobes. Op, operculum. In, Island of Reil. Cls, claustrum. f''', third frontal convolution. *Th*, thalamus, NC, caudate nucleus; NC', tail of caudate nucleus. LN, lenticular nucleus. 1, 11, 111, first, second, and third divisions of the lenticular nucleus. EK, external capsule. IK, posterior division; IK', anterior division; and K, knee of the internal capsule. *ah*, *ph*, anterior and posterior horns of the lateral ventricles. *gcc*, knee of the corpus callosum. *sp*, splenium. *mc*, middle commissure, *f*, fornix. *sl*, septum lucidum. *a*, cornu Ammonis.

* To obtain this view Flechsig makes a horizontal section through the brain, just above and parallel with the Sylvian Fissure.

The internal capsule is, in fact, composed of two segments, the one anterior, the other posterior, the two being united by what Flechsig terms the knee of the capsule (Fig. 21, K). It is now known that the fibres of the pyramidal tract are contained almost wholly in the anterior two-thirds of the hinder segment of the internal capsule (Fig. 21, IK′).

We have spoken above of the descending degenerations resulting from lesions of the anterior segment, and of the genu of the internal capsule, neither of which seems to be traceable below the pons or medulla. It remains, therefore, to speak of degenerations due to lesions of the posterior segment of the internal capsule, or of parts of the hemisphere just above in relation therewith.

As we have already shown, lesions of the posterior third of the hinder segment of the internal capsule give rise to no descending degenerations, though they occasion cerebral hemianæsthesia (p. 152.)

There is, in fact, abundant evidence to show that this portion of the internal capsule is composed of afferent rather than of efferent fibres ; so that if secondary degenerations exist at all, as a result of lesions of the fibres passing through this region, they should be ascending degenerations—of which, however, nothing is at present known.

It seems equally clear, moreover, that the great bulk of the pyramidal fibres issuing from the Rolandic area of the cortex become massed together in the anterior two-thirds of the hinder

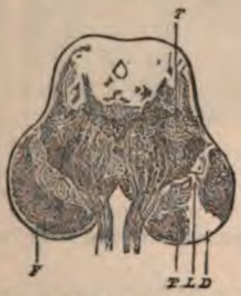

Fig. 22. Transverse Section of the Peduncular Region in a case of Secondary Degeneration [after Charcot].

T, upper layer of tegmentum lying beneath the anterior quadrigeminal bodies. F, lower layer or foot on healthy side. L, locus niger P, internal fasciculus of lower layer on diseased side. D, secondary degeneration occupying about the middle two-fourths of the foot of the crus.

Between the two peduncles are the third nerves, passing upwards to their nuclei beneath the Sylvian aqueduct.

segment of the internal capsule. The remainder of them are situated in the contiguous genu. As already mentioned, these latter descend only as far as the pons and the bulb. The great bulk of the pyramidal fibres, however, contained in the hinder segment descend into the spinal cord, after passing through regions, now to be described, in the cerebral peduncle, the pons, and the bulb.

The part of the cerebral peduncle which is implicated in association with the anterior two-thirds of the hinder segment of the internal capsule is the middle third or two-fourths of its lower layer, commonly known as the 'foot.' This part is separated from the upper layer or 'tegmentum' by the well known pigmented layer, or 'locus niger.'

Fig. 22 is intended to show the area occupied by the band of secondary degeneration in the upper part of the cerebral peduncle in a well marked case, before shrinking has set in, and as it appears on a transverse section; while Fig. 23 is intended to illustrate the fact that when such a degeneration is old and well marked, an external examination of the crus, pons, and medulla, will show evidences of an

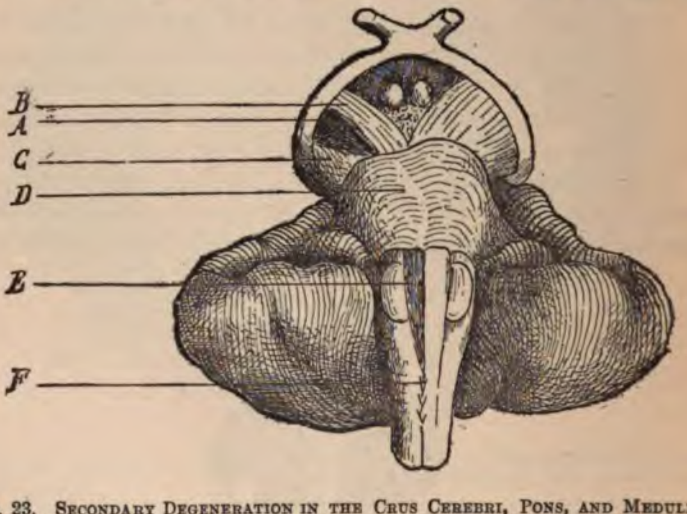

FIG. 23. SECONDARY DEGENERATION IN THE CRUS CEREBRI, PONS, AND MEDULLA [after Charcot].

A, Degeneration in the crus cerebri (pyramidal tract). B, Internal peduncular fasciculus degenerating under occasional, but exceptional, circumstances. C, External fasciculus (? centripetal) never degenerating. D, Pons Varolii (it is asymetrical, being atrophied on the side of the lesion). E, Greyish, atrophied, and degenerated pyramid, decussating with its fellow at F.

atrophic change. In the crus and medulla, where the altered parts appear at the surface, they present also a more or less distinct greyish tint. A few details concerning the disposition of the tract of degeneration in these different regions may prove useful.

In the *crus cerebri*, the degenerated area appears at the surface in the form of a triangular space, situated in the middle region of the lower layer or foot (Fig. 23). The base of the triangle is above, and its apex next the pons. The surface of the foot of the peduncle is thus divided into three regions:—(*a*) the middle region, represented by the degenerated pyramidal tract; (*b*) the external region, which, according to Charcot's numerous observations, never becomes the seat of degeneration; and lastly (*c*) an internal region, which only under very exceptional circumstances becomes the seat of degeneration, though when it does, such degeneration may exist alone, that is, without the median tract participating in the same change.

When a transverse section of the peduncular region is made, the ordinary degeneration in the pyramidal tract is seen in the form of a rudely triangular space (Fig. 22, D) occupying the middle part of the foot, and extending from the locus niger above, downwards to the surface of the crus. The superficial surface is wider than that next the locus niger.

In the *pons* some amount of flattening and diminution of size occurs on the side of degeneration, when this happens to be well marked and old, but there is no other external indication of the existence of a secondary degeneration. Within the substance of this region it is, moreover, by no means so easy to recognize and define the degenerated tracts when transverse sections are made. On this subject Charcot says:—" In the pons, the pyramidal tract is dissociated and extremely difficult to recognize in the midst of the transverse fibres peculiar to the part. Nevertheless the secondarily degenerated fasciculi may still

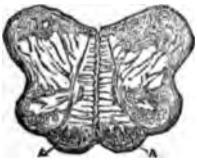

FIG. 24. TRANSVERSE SECTION OF THE MEDULLA OBLONGATA ON A LEVEL WITH THE MIDDLE OF THE OLIVARY BODY, SHOWING (A, A) SCLEROSIS OF BOTH ANTERIOR PYRAMIDS [after Charcot].

In Hemiplegia the degeneration occurs only on one side, rather than in both anterior pyramids as it did in this case of 'insular sclerosis.'

be distinguished fairly easily, especially on comparison with the healthy side, in the lower or bulbar regions of the pons, where these fasciculi begin to collect and once more form into groups preparatory to constituting the pyramidal tract a little lower down."

Throughout the *medulla oblongata* the secondary degeneration may be recognized with ease. The anterior pyramid on the affected side is seen to be smaller, flatter and greyer than its fellow; and in transverse sections made through the medulla the degenerated tract may be seen to be strictly limited to the region of the pyramid.

What has been said previously may be summed up in the following manner :—

(1).—Lesions limited to the anterior segment of the internal capsule, which is presumed to be largely composed either of a special set of fibres proceeding from the Rolandic area or else of certain fibres emanating from the cortex of the frontal lobe, cause secondary degeneration, but this degeneration does not involve the pyramidal tract proper; it shows itself by a grey band occupying the inner third of the foot of the peduncle (Fig. 19). In reference to the fibres entering into this tract Charcot says :—" It is very probable also that these fibres are arrested below at some point in the pons, for when this fasciculus is degenerated it is impossible to detect it in the corresponding pyramid; *à fortiori* it does not pass down into the spinal cord."

(2).—Lesions limited to the knee of the internal capsule (Fig. 20, D) cause the degeneration of a slender fasciculus, named by Brissaud the 'geniculate fasciculus,' which descends in the foot of the peduncle between the internal and the middle third of this body. This fasciculus contains efferent fibres proceeding from the region of the third frontal convolution to nuclei in the medulla oblongata, which are probably concerned with movements of the lips, tongue, soft palate, and larynx. This geniculate fasciculus is sometimes found degenerated in old cases of aphasia.

(3).—Lesions limited to the anterior two thirds of the hinder segment of the internal capsule, cause secondary degenerations which descend in the middle third of the foot of the peduncle (Fig. 23) and thence into the anterior pyramid of the same side. This portion may, therefore, be called the pyramidal region of the capsule, since the nerve fibres which traverse it seem to be continued directly into the pyramidal tracts, through which the muscles of the opposite limbs and of the trunk of the body are innervated.

(4).—Lesions in the posterior third of the hinder segment of the internal capsule, give rise to no descending degenerations. This region appears to contain only afferent fibres, which come to it from below through the outer third of the cerebral peduncle—a tract in which descending degenerations are never seen. Destructive lesions in this region of the cerebral peduncle and internal capsule ought, indeed, to give rise to ascending degenerations, though they have not yet been observed. Circumscribed lesions in this region produce that combination of symptoms which has been previously described under the name of cerebral hemianæsthesia (see p. 152).

In regard to the ultimate destination of the efferent fibres proceeding, in the manner above defined, from the Rolandic area of the cortex, it is now desirable to say a few words.

Some pass downwards as far as the lumbar swelling of the spinal cord, and there, in all probability, terminate in some of the large motor cells in the anterior cornua from which issue nerve fibres for the lower extremities. Others pass down into different segments of the dorsal region of the cord, there to come into relation with ganglion cells in the anterior cornua, and through these to influence nerve fibres proceeding to the muscles of the trunk. A very large number of fibres, however, go no further than some part of the cervical swelling of the cord; they terminate there, principally, if not wholly, in the anterior cornua, and thus come into relation with the numerous nerves proceeding to the muscles of the upper extremities. Other fibres stop at higher segments of the cervical portion of the cord, and others still in the medulla and in the pons—in each region coming into relation with ganglion cells, either in the anterior cornua or in related parts, and, through them, influencing motor nerves which proceed to the neck and some trunk muscles, the diaphragm, parts of the alimentary tract, the larynx, the bronchi, the facial muscles, and the ocular muscles.

It must be evident, therefore, that from the level of the pons downwards the sectional area of any tracts of secondary degeneration should go on diminishing, through the medulla and spinal cord, owing to the constant termination of sets of the degenerated fibres in successive regions, as above indicated.

Whether some of the fibres from the cortex stop short in the corpus striatum, and whether others pass downwards from the cortex into the pons and thence upwards through the middle peduncle to some portion of the cerebellum, are problems concerning which we have no definite knowledge at present, but they are, nevertheless, of extreme importance with a view to the full understanding of the mode of action of the cerebrum and the cerebellum in the execution of muscular contractions of all kinds.

Formerly, it was supposed that the peduncular motor fibres emanated from the corpora striata, in which they were in relation with ganglion cells; and that an entirely different set of fibres connected the cortex with the corpora striata, through the intervention of which, it was believed, all voluntary movements were executed. Since the date of the experiments of Hitzig and of Ferrier, numerous pathological observations have been made which have clearly shown that destructive lesions of the Rolandic area alone, and independently of all structural damage to the corpus striatum or to the internal capsule, give rise, as already stated, to typical secondary degenerations which take the ordinary course downwards, and end in the usual manner in the lumbar region of the spinal cord. All the fibres that undergo degeneration in this way, must, clearly, belong to the category of what have been termed 'direct peduncular fibres.' On the other hand, it must not be too hastily inferred that all the efferent fibres from the Rolandic area belong to this category. Some of them, as they are passing between the caudate and the lenticular nuclei may easily pass into one or other of these bodies; and yet it may be very difficult, nay impossible at present, to offer any proof that such is the case. How difficult would it be, in the case of any such lesion, definitely to affirm that the number of degenerated fibres in the crus cerebri were just equal to those in the foot of the corona radiata. If we look to the different mode of aggregation of the fibres in these two sites it might well be that, in the crus the number of such fibres may have diminished even to the extent of one-fourth, and yet the fact of such diminution might escape all positive observation.

The modern point of view is the reverse of that which formerly obtained in regard to the relative importance and number of the 'direct' and the 'indirect peduncular fibres.' The presence of a large proportion of fibres belonging to the former category is now established beyond doubt. The existence of those of the latter category is at present doubted, and however probable their presence may be it cannot be actually demonstrated. As we have suggested above, the existence of efferent fibres from the Rolandic region terminating in the caudate and lenticular nuclei, may be real and yet may easily escape actual observation. The presence of some such fibres would be an almost necessary corollary of the existence of any large number of 'indirect peduncular fibres' (that is to say, of efferent fibres taking their origin from the corpus striatum, and, therefore, having only an 'indirect' relation with the cerebral cortex). If such fibres exist, then it would seem, at first sight, that extensive lesions limited either to the caudate or to the lenticular nucleus ought to produce some amount of secondary degeneration in the peduncular tracts below. But it has been definitely asserted by many excellent observers that such lesions give rise to no secondary degenerations. Thus, in reference to this point, Charcot says:—"Lesions which remain limited to the substance of the central grey masses, viz., the caudate and lenticular nuclei and the optic thalamus, do not give rise to secondary sclerosis. This remarkable fact was clearly demonstrated by L. Turck in 1851. M. Vulpian and myself have confirmed its perfect accuracy by the investigations which we made in common on this subject at La Salpêtrière from 1861 to 1866. M. Bouchard's important labours have likewise corroborated it."

The fact of the absence of degenerations as a result of lesions in these grey nuclei would seem, therefore, to be extremely well attested, and the question naturally arises whether it is possible to reconcile such a fact with what we feel to be the great probability that indirect peduncular fibres do nevertheless exist. We think this may be done, and some further facts that have been ascertained in reference to lesions of the caudate nucleus may guide us to such a reconciliation. Charcot says:—"I must add that, according to all probability, a supplementary relation may be established not only between the various grey nuclei, but even between different parts of the same nucleus. It has been shown at any rate as far as the caudate nucleus of the corpus striatum is concerned, that partial destructive lesions affecting the most diverse regions of the nucleus are uniformly indicated by hemiplegia which, although transitory, is more or less pronounced and total, that is to say involving at once both the face and the extremities. In this respect there is no difference to be noticed between the head, tail, and middle part of the caudate nucleus. Accordingly it appears, as Dr. Hughlings Jackson has justly remarked, that each particle of the corpus striatum is a miniature representation of the entire corpus striatum. Experiment, moreover, yields results in conformity with those furnished by clinical observation, in showing that stimulation of parts of the caudate nucleus, however made, always produces combined movements on the opposite side of the body, and never dissociated movements, localised, for example, to an extremity or to part of an extremity."

From the facts above referred to, and also from what is known as to the histological structure of the caudate and lenticular nuclei, it would seem probable that the relation between the nerve cells in the caudate and lenticular nuclei, and any efferent fibres issuing from these ganglia, would be quite different in kind from that existing between the efferent fibres in the cortex of the Rolandic area and the large nerve cells there situated. In the latter case the relation is of a very definite nature, owing to the continuity of one particular process from the base of the cell with the axis cylinder of an efferent nerve. In the case of the corpus striatum, however, the facts seem to suggest multiple relations of the ganglion cells with one another and with the efferent fibres, and the probability that the fibrils of the efferent nerves come into relation with plexiform networks in possible connection with large numbers of ganglion cells. The existence of some such anatomical connections might go far to account for the absence of definite secondary degenerations as a result of lesions limited to either one of the ganglia of the corpus striatum.

Experimental observations on monkeys may, perhaps, throw light upon this subject in the future. Thus, it would be important to compare with great exactness the size and precise distribution of the sectional areas of tracts of secondary degeneration in different parts below the internal capsule, resulting (*a*) from destruction of the whole of the Rolandic area of the cortex with those (*b*) to be found in corresponding situations in other animals in whom a section had been made through the lowest part of the internal capsule. In the former case we should have to do with degeneration of the direct peduncular fibres only; whilst in the latter case, the area of degeneration might be larger and somewhat differently disposed, because of the possible existence in this case not only of degeneration of the direct, but also of the indirect peduncular fibres. The latter class of fibres, if any such exist, would almost certainly degenerate if severed after leaving the corpus striatum. For, in that case, such fibres would be as much severed from any ganglionic influences needful for their nutrition as if their relation with ganglion cells had been single and direct, as it is with cells and efferent fibres in the Rolandic area of the cortex. It is very important that this matter should be cleared up.

In the *spinal cord* the degenerated pyramidal band divides into two portions corresponding with the situations of the direct and of the crossed pyramidal tracts—the latter being, in the great majority of cases, by far the larger of the two (see p. 161), and situated in the posterior part of the opposite lateral column, whilst the direct pyramidal tract continues along the inner part of the anterior column of the same side.

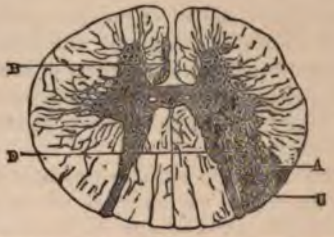

FIG. 25. DESCENDING SECONDARY DEGENERATIONS IN THE SPINAL CORD, COEXISTING WITH A RIGHT SIDED HEMIPLEGIA [after Charcot].

Transverse section of the cord in the cervical region. A, degeneration of the crossed pyramidal tract in the right lateral column. B, degeneration of the direct pyramidal tract. C, area of white matter corresponding to the direct cerebellar tract. D, region intermediate between the posterior cornu and the pyramidal tract, which is never affected in descending degeneration.

These two tracts of degeneration are to be traced throughout nearly the whole length of the cord into the lumbar region, diminishing gradually in size and also varying slightly in shape and disposition in the different regions. Other portions of the cord, and also the nerve roots on the affected side, show no sign of any change.

It was stated by Bouchard, and has been commonly repeated by succeeding writers, that the direct pyramidal tract in the anterior column wears itself out, and does not appear beyond the mid-dorsal region. This, however, is by no means always the case. As the writer pointed out in 1867, such bands of degeneration may, like those of the crossed pyramidal tracts, not disappear till the lower part of the lumbar swelling is reached.

The area of the degenerated tract in the anterior column also varies much, in different cases, in accordance with variations in the proportion of the decussating and the non-decussating fibres of the pyramidal tract in different persons.

Transverse sections made through the cervical enlargement (A) show an area of degeneration in the lateral column having the form of a triangle with distinctly defined margins, the summit of which is directed inwards towards the angle separating the anterior from the posterior grey cornua, whilst its base, somewhat rounded off, is always separated, both from the periphery of the cord and from the outer aspect of the posterior cornu, by layers of undegenerated white substance.

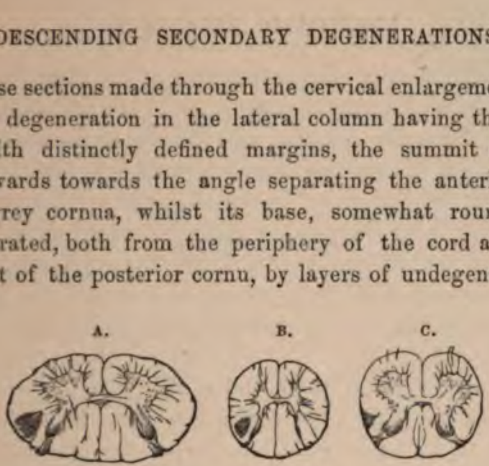

FIG. 26. TRANSVERSE SECTIONS OF THE SPINAL CORD SHOWING SECONDARY DEGENERATIONS ON THE LEFT SIDE, AS A RESULT OF CEREBRAL SOFTENING WHICH HAD DESTROYED THE OPTO-STRIATE BODIES AND THE INTERNAL CAPSULE IN THE RIGHT HEMISPHERE [after Charcot].

A, cervical region. B, dorsal region. C, lumbar region.

In the dorsal region (B), the degenerated area gradually diminishes in its diameter and tends to assume an oval form.

Lastly, in the lumbar region (C), the degenerated area is again of a triangular shape, though its base is now quite superficial and lies immediately beneath the pia mater. It becomes superficial here because the 'direct cerebellar tract,' which in higher segments of the cord lies next the surface, does not exist in the lumbar region. (The fibres of the direct cerebellar tract are supposed to take their origin from the cells in the vesicular column of Clarke, which only begins to make its appearance in the upper part of the lumbar region. This and other ascending degenerations will, however, be considered in another portion of this work—see Part IV, Paralyses due to Lesions of the Spinal Cord).

Thus we find that **descending secondary degenerations** are caused by lesions of a destructive character, only when they occupy certain limited and well defined regions in the brain. These regions extend from the cortex downwards and inwards in each cerebral hemisphere, in the form, roughly speaking, of two much elongated cones the apices of which come into contact in the medulla elongata. Their bases are situated in the Rolandic areas, in which are comprised, on each side, (1) the ascending frontal and the ascending parietal convolutions, (2) the posterior extremities of the three frontal convolutions, (3) a part of the parietal lobule, and (4) a part of the marginal convolution. From this cortical area the region narrows downwards to certain parts of the internal capsule already defined. Lower down, it narrows still further in the foot of the crus cerebri; whilst below this part, there is, in the pons, a slight widening out in the area of distribution of the efferent tracts, followed by a condensation and gradual approximation of the pyramidal tracts of the two sides as they pass through the lower part of the pons and the upper part of the medulla oblongata.

Below the upper part of the medulla, the pyramidal tract of each side bifurcates unequally, the larger and most important segment decussates with its fellow ('decussation of the pyramids') so as to gain the hinder part of the opposite lateral column of the cord (crossed pyramidal tract), along which it can be traced into the lumbar region of the cord; whilst the smaller and less important segment (direct pyramidal tract), continues through a considerable extent of the spinal cord, occupying the inner part of the anterior column on the same side as the brain lesion.

XX. THE CAUSATION OF CONTRACTURE, ANKLE CLONUS AND EXAGGERATED KNEE-JERK; AND THE EXTENT TO WHICH THEY ARE DEPENDENT UPON CEREBELLAR INFLUENCE.

After this study of the secondary degenerations which occur in the brain and the spinal cord (throughout the pyramidal tracts), we shall be the better prepared to seek some solution of the extremely difficult problem as to the mode of origin and real meaning of late rigidity, and what we have now ascertained to be its invariable accompaniments, viz., more or less exaggerated knee-jerk and ankle clonus.

In the first place, it is essential to point out that precisely the same set of phenomena occur when the lateral columns of the cord are the seat of a primary rather than a secondary sclerosis.

Then again it would seem that the condition of the cord and of the limbs, leading to the manifestation of exaggerated knee-jerk and ankle clonus, constitute the beginning of a state which, when it happens to be, or to become, developed to a higher degree, is certain to lead on to the appearance of the condition known as 'late rigidity' (Brissaud, 'Rech. sur la Contracture permanentes des Hemiplégiques,' 1880.)

It is maintained by Brissaud and others, that the early stages above referred to, and also those later stages in which contracture becomes more and more developed, are conditions in which there is, at first, merely a slight exaggeration of that state of the muscle which is spoken of as its tonus, and that as the spinal or cerebro-spinal condition above referred to becomes more and more developed, so does this particular state of the muscle become more and more marked. There is much to be said in favour of this view. It is well known that such contractures may exist for a very long time—that is, for years—and yet, under the influence of chloroform, the muscles may all completely relax in such a manner as to show that no local organic changes or retractions are to be regarded as the causes of the previously habitual state of firm rigidity. Brissaud has also ascertained that the contracture may be made to disappear in another very simple manner, namely, by the application of an Esmarch's bandage. This, by rendering the limb bloodless, at the same time deprives the muscles of their contractility. After the bandage has been applied for about twenty minutes, the deformity of the limb begins to diminish, and shortly the condition of rigidity (which had, perhaps, existed for years) may have completely disappeared. But, as Brissaud says, "immediately that the bandage is loosened and the circulation becomes re-established, the deformity reappears in the course of a few seconds." In only two, out of a very large number of old cases of hemiplegia with contracture at the Salpêtrière, did Brissaud discover any evidence of actual organic changes in the joints.

It may now, therefore, be considered as generally admitted (*a*) that there exists, in association with sclerosis of the lateral columns of the cord, whether primary or secondary, an exaggeration of the muscular tonus; (*b*) that this state of the muscle and cord favours an exaggeration of the knee-jerk, or (in its more developed condition) leads actually to the manifestation of contracture; and (*c*) that even in the most extreme forms of contracture we have to do with persistent muscular contraction differing, except for its persistence and mode of origin, only in certain unimportant respects from that which occurs as a result of volitional incitations.

We may take these positions to be admitted by those who now seek to explain the cause and mode of production of the state known as contracture. It would be useless to consider seriously the old explanations advanced by Todd and others; we must restrict ourselves to the explanations based upon the more modern point of view, that contracture is essentially related to lesions which damage the motor tract, or else to functional perturbations which render portions of this important tract functionally inactive. The notions that have been advanced hitherto, are chiefly the four now to be referred to.

(1). Hitzig is inclined to regard the continuous contracture of hemiplegics as a manifestation of repeated 'associated movements,' favoured partly by a diffusion of volitional impulses, and partly by an excited or unduly irritable condition of certain motor centres, caused in some way by the original brain lesion (p. 183). This view seems obscure and extremely difficult to understand, seeing that the contracture exists, not only at times when movements are being executed, but also during periods of rest, and even during the long hours of sleep. The fact that the contracture is aggravated during the waking state, and is always increased when movements are attempted, must, therefore, be explained in some different manner.

(2). Others, relying upon the fact that, in some of the lower animals, the reflex activity of the spinal cord seems to be greatly exaggerated after a section has been made through one of its higher segments, so as to sever it completely, from the brain, seek to explain hemiplegic contracture by supposing it to be due to the cutting off, from certain parts of the spinal cord, of the moderating influence of the cerebral cortex, owing to the presence of a lesion involving some part of the pyramidal tract. This explanation alone is evidently not adequate. If it were so, we should have the condition manifesting itself immediately on the occurrence of a cerebral lesion which suffices to cut off cerebral influence from one side of the spinal cord, which is not the case. Further evidence against the adequacy of this explanation will also appear directly.

(3). Hughlings Jackson very properly lays much stress upon this cutting off of the cerebral influence, believing it to be a permissive condition, allowing a then unantagonized cerebellar activity to manifest itself. He advances this as a mere tentative hypothesis ('Med. Times and Gaz.,' Feb. 12, 1881), without supporting it by much positive evidence. We shall see, however, directly, that much may be said in favour of some such explanation.

(4). We now come to the view that has been advocated by Bouchard, Charcot, Brissaud, and others—it is, in fact, essentially the doctrine of the French school. They do not take adequate cognizance of the fact that, in cases in which late rigidity is to appear, there is almost invariably, from the first, the minor degree of this condition, as evidenced by the existence of an exaggerated knee-jerk and the presence of ankle clonus. Brissaud is certainly in error when he says in reference to this point (*loc. cit.* p. 129) "il est certain que les caractères morbides des réflexes musculaires ne se révèlent jamais qu'à partir de la formation de la sclérose latérale." Bouchard intimates that the mere cutting across of the lateral column gives rise to a paralysis with flaccidity, and that when the paralysis becomes associated with contracture, this is to be considered as "an effect of the sclerotic myelitis of these columns, which occurs in the course of their secondary degeneration." This point of view is insisted upon, and elaborated by Brissaud, who points out that the fibres of the degenerating column are, in their healthy state, in immediate functional relation with the great nerve cells in the anterior cornua, and he assume that the irritation of these fibres by the overgrowing neuroglia, would necessari y keep up a continuous condition of irritation in the ganglion cells, and thus tend permanently to maintain the signs of contracture, which is, as he thinks, a direct result of this over-excitation of the motor cells of the anterior cornu. But some positions are assumed here which are far from being proven—are, indeed, rather improbable. Where the nerve fibres have become degenerated, it must not be too readily assumed that the axis cylinders of such nerves are able to become irritated in the manner supposed, and likewise able to transmit such irritation to the ganglion cells. It is true that healthy fibres between, or around those which are degenerated, might become stimulated in this manner, but this would be the stimulation of fibres still in relation with the cerebrum. Again, the fact of the existence of a form of contracture in hysteria, exactly comparable with that which may be met with in hemiplegia or paraplegia, also tells decidedly against the doctrine advocated by Brissaud, since he does not venture to postulate the existence of any irritative lateral sclerosis in these cases. I have, indeed, seen rigidities of both lower extremities, with marked ankle clonus and greatly exaggerated knee-jerk, existing for many weeks in a young girl suffering from Pott's disease, and when death took place from an intercurrent lung affection it was found, on most careful examination, that although there had been pressure on the antero-lateral columns, there was no trace of descending degeneration to be found. This was verified by a careful microscopical examination.

The view of Brissaud and of Charcot, in short, assumes that the effects in question are explicable on the supposition that there is an increase of the condition known as tonus; that this condition implies an unnatural state of activity in the ganglion cells of the anterior cornua; and, moreover, that such a continuous increase of functional activity can be evoked by a continuous morbid stimulus in the spinal cord itself—stopping short of being excessive, because an excessive stimulus of this same kind is appealed to, by the same pathologists, as the cause, when it occurs, of the occasional wasting of the ganglion cells together with an atrophy of the related paralysed muscles and the disappearance of all contracture (p. 233).

I am inclined to adopt a view which is related in part to the last three mentioned, but which is most closely allied to the notion of Hughlings Jackson. We may accept the general view of the French school, as to the fact that the symptoms themselves may be caused by an exaggeration of muscular tonus, and yet we need not accept their view as to the mode in which the nervous conditions that underlie, or cause, this exalted tonus are generated. If we suppose, for instance, that the condition known as muscular tonus is mainly due to cerebellar influence acting upon and through the spinal centres, then, it may well be, that the removal of cerebral influence from certain parts of the spinal cord may allow cerebellar influence to reach such parts of the cord much more freely than natural—that is, as H. Jackson says, we should have to do with an unantagonized, or as I would rather say an unrestrained, influx of cerebellar energy. If, therefore, the extra irritability in certain portions of the grey matter of the cord, of which we have evidence in cases of disease of its lateral columns, be due to certain influences, in excess of what is customary, reaching it from the cerebellum, some evidence ought to be forthcoming to this effect. Let us see what facts can be brought forward to elucidate this question, and in support of my position generally :—

(a).—That the suppression of the cerebral influence is an important factor in leading to the increased irritability or over-action of muscles, which we are now seeking to explain, is evidenced by a recognition of the muscles which are affected and of those which escape. Those chiefly affected are the muscles most paralysed in cases of hemiplegia ; whilst those which escape are the very muscles that are not paralysed in cases of hemiplegia—viz., the muscles of the trunk and others that act bilaterally. But Broadbent's hypothesis is applicable in the one case as it is in the other. If the bilaterally-acting muscles do not manifest rigidity, it is because they are not really cut off from cerebral influence, owing to the spinal nuclei in relation with these muscles being so closely connected by means of commissural fibres—and, therefore, being open to innervation on one or the other side, through the particular cerebral hemisphere and pyramidal tract that may remain intact.

(b).—We may admit the fact that where there is a suppression of the cerebral influence, there is an increase of muscular tonus or irritability, without necessarily supposing, as Bouchard and Charcot do, that this increase in the muscular irritability is dependent only upon conditions having their origin within the spinal cord itself.

(c).—The fact that such muscular irritability, in patients suffering from slight contracture, is increased if they take strychnia, has been commonly held to prove that this irritability is dependent upon changes or conditions existing within the spinal grey matter alone. But if we bear in mind that the muscular irritability in such cases is similarly exalted by mental activity or excitement, or by the performance of voluntary movements, and that it is often notably diminished by sleep, we may see, in these facts, reasons for believing that the excitability of the grey matter of the cord increases or diminishes with the excitement or the reverse of some encephalic centres, and that an excessive influence of some kind, producing increased tonus in the paralysed muscles, must reach the related ganglion cells of the spinal cord through other channels than the damaged pyramidal tract.

(d).—The notion that the increased excitability of certain spinal centres, in cases where cerebral influence is more or less completely cut off therefrom by the existence of lateral sclerosis, is due to influences operating upon them from some other encephalic centre, is further borne out by the fact that when in cases of spinal cord disease partial lesions have at first existed (associated with rigidity and great exaltation of the deep reflexes), and this primary condition of the cord has been followed by a total transverse softening, then the clinical picture has been completely changed at once. The limbs become completely flaccid, ankle clonus wholly disappears, and the knee-jerk also is lost (Quain's 'Dictionary of Medicine,' p. 1480). This I have now seen in three cases, each of which was under observation for many weeks, and in each an autopsy was subsequently made which showed the existence of a total transverse softening implicating the upper or mid-dorsal region for a length of one or two inches. What could be the cause of the complete change in the clinical picture in these cases, if we are not to suppose that it was due to the cutting off of *some* encephalic influence which previously reached the grey matter of the cord below the seat of lesion?

Precisely similar evidence is derivable from cases in which lesions of certain kinds are situated within the encephalon. Thus I have recorded a case of apoplexy due to a rapidly established thrombosis of the basilar artery ('Trans. of Clin. Soc.,' 1885), in which, within one hour from the onset of the attack, there was a generalised paralysis of the body together with a complete absence of all reflexes both superficial and deep. Now a case of this kind presents us with a sort of natural experiment, closely equivalent to an experimental section made between the pons and the medulla oblongata. Besides cutting off the cerebral motor influence from both sides of the cord, its effect would also be to paralyse the influence exerted by the cerebellum upon the spinal cord through its middle peduncles—which are the parts probably containing the great bulk of its outgoing fibres. This would have been the kind of case in which, according to commonly accepted physiological teaching deduced from cases of experimental section of the cord in lower animals, the spinal cord ought to have shown an exaggeration of its ordinary reflex functions. Yet, as a matter of fact, all the ordinary spinal reflexes were abolished. Why was this so? The answer is, I believe, in great part to be found in the fact that the nervous system is more evolved in man, and nervous symptoms are correspondingly more differentiated, so that functions which are performed in many of the lower animals by the aid of the spinal cord come to be largely dependent in man upon the cerebellum. Consequently it is unsafe to argue that effects following section of the spinal cord in such lower animals will repeat themselves as a result of equivalent conditions occurring in man. Again, evidence drawn from more ordinary apoplectic conditions, due, for instance, to large hæmorrhages in one or other cerebral hemisphere, suffices to illustrate the same position. Thus, by way of example, I may say that a few weeks ago I had the opportunity of observing the following phenomena in a case of ingravescent apoplexy (p. 157), shown post-mortem to be due to a large hæmorrhage into the middle of the substance of the right hemisphere. During the first two days, with the existence of a complete left hemiplegia and a condition of well marked stupor, no knee-jerk and no ankle clonus was to be obtained on the paralysed side, whilst on the right side the knee-jerk was normal and there was no ankle clonus. Two days later the paralysis, with complete flaccidity of limbs, had spread so as also to involve the right side of the body; coma was profound, and there was now an absence of all reflexes, both superficial and deep, on the right as well as on the left side. So far as the spinal functions were concerned, this case, in its later stage, presented effects precisely similar to those met with in the case of thrombosis of the basilar artery. At the autopsy, also, there was evidence, owing to the amount of the bleeding and other causes, of a great increase of pressure within the cranium. The convolutions over both hemispheres, but especially over the right, were notably flattened, and this could scarcely have existed without entailing an almost corresponding amount of pressure upon the pons Varolii. Here again, then, at the close there may have been a withdrawal not only of cerebral but also of cerebellar motor influence from the cord, a condition which, as in the last case, coincided with an absence of all reflexes. There seems to be a uniformity in the testimony supplied by these different cases.

What has been said above suffices to show, in my opinion, an extreme probability that it is the cutting off of the cerebellar influence which produces the abolition of the deep reflexes in the later stages of the cases cited. Conversely, I believe that the maintenance of ordinary cerebellar relations with the spinal cord is an essential condition of normal tonus in muscles; and also that the signs of exaggerated muscular tonus (viz., exaggeration of deep reflexes, without or with the presence of muscular rigidities) associated with secondary or primary sclerosis of the lateral columns of the spinal cord, may be due to the mere cutting off of cerebral influence from corresponding parts of the spinal cord, and to the consequent unrestrained influence (in excess) of the cerebellum upon these parts of the cord. Excessive cerebellar influences reaching the anterior cornua of the spinal cord, seem to determine therein an increased irritability, which shows itself by an exaltation of muscular tonus, or, under more extreme conditions, by the production of actual rigidity of muscles. In my opinion, the weakening or removal of cerebral influence from the spinal cord leads to the weakening or removal of an inhibitory influence which (operative probably in the pons Varolii) usually regulates or restrains the outflow of cerebellar energy through its median peduncles. I would not, in the present state of knowledge, attempt to define in what precise way the cerebrum and the cerebellum co-operate with one another in their possible actions upon the different muscles of the body (see 'Brain as an Organ of Mind,' pp. 503-510). In the performance of the most automatic actions the cerebellum may come into play to a considerable extent independently of the cerebrum, and such neuro-muscular processes are comparatively little interfered with by unilateral lesions of the cerebrum. In the performance of the least automatic actions, however, the cerebrum altogether takes the lead, and the cerebellum acts only as it is solicited or permitted to act, in directions indicated by the outgoing cerebral incitations. The withdrawal, owing to unilateral lesions, of cerebral influence from muscles which are principally called into action voluntarily, is, therefore, well calculated greatly to interfere with 'the balance of power' usually capable of being brought to bear upon such muscles, and may lead (as it seems to do) to their being acted upon in excess by the cerebellum, even when in a state of rest—in consequence of which there is increased tonus, carrying with it exaltation of deep reflexes or even muscular rigidities.

We may as a working hypothesis adopt such views as I have above attempted to sketch, even though we know nothing definite, at present, as to the precise channels by which this cerebellar influence reaches the anterior grey matter of the cord. That is a point which must be solved by future researches. What has been said is quite in accordance with the now commonly received views as to the exact mode of production of knee-jerk and of ankle clonus. The view that they are true reflexes, as at first supposed, has been nearly abandoned. They are immediate results of the mechanical excitation of tendons, and yet, in order that such results should follow such causes, a certain increased degree of tonus in the muscle is necessary. Muscular tonus is supposed to be maintained by a kind of reflex influence emanating from the muscle itself; the result (that is, the amount of tonus manifested) being as we have above endeavoured to show, dependent upon the nature of the connections existing between the anterior spinal cells and the two great encephalic centres, as well as upon the structural and functional integrity of these spinal cells themselves. It is well known that disease, either of the ingoing or of the outgoing fibres to or from the spinal centres, will interfere with the maintenance of a proper muscular tonus, and will, as a consequence, render the manifestation either of ankle clonus and even of the knee-jerk impossible under the influence of its usual stimulus.

Why the greatly increased freedom of the knee-jerk, and the manifestation of ankle clonus with more or less of rigidity of muscles, should, as a rule, manifest themselves rather slowly and not immediately follow the onset of the cerebral lesion, when it is of such a nature as to cut off the cerebral motor influence from certain regions on one side of the spinal cord, is a point not at all easy to explain. It may be that in such cases the extra leakage of cerebellar energy, which the cerebral lesion permits after the shock occasioned by its occurrence has had time to resolve, has a tendency to go on increasing up to a certain point, because of the gradually lessening resistance (probably in the pons) opposed to any such overflows of cerebellar molecular energy. All nerve actions, whether normal or abnormal, become easier, and recur all the more readily, the more frequently they are repeated.

Although, in its general aspects, my view is very similar to that first enunciated by Hughlings Jackson, yet I differ from him much in details as to the manner in which the cutting off of the cerebral influence leads to the relatively greater influence of the cerebellum upon the muscles involved. I cannot, for instance, believe with him that the motor cells in the spinal cord are commonly the seat of antagonizing activities emanating from the cerebrum and the cerebellum respectively, and that the explanation of the exaggeration of deep reflexes, in the cases we have been considering, is to be found simply in the withdrawal of one member of an antagonizing couple usually operative upon the spinal motor cells ('Examiner,' Ap. 5, 1877).

It is true that in many respects, also, the views at which we have arrived concerning the functions of the Cerebellum are in accordance with one another. And yet our points of departure are wholly different. His general conception seems to be that all the muscles of the body are capable of being influenced, though to an unequal extent, both by the cerebellum and by the cerebrum; that the cerebellum exercises the most influence over the muscles which are called into play in the most automatic movements, and the cerebrum, on the contrary, over the muscles most frequently called upon to produce voluntary and frequently changing movements. He seems to be content to explain this in accordance with the somewhat metaphysical hypothesis put forward by H. Spencer ('Principles of Psychology,' Vol. I, p. 61) that "the cerebellum is an organ of doubly compound co-ordination in space; while the cerebrum is an organ of doubly compound co-ordination in time"—supposing the common function of the two to be "that of co-ordinating in larger groups and in various orders, the impressions and acts co-ordinated in the lower centres." This view assumes, therefore, an original division of the field of functional activity in regard to movements between the two organs, which, to a considerable extent, coincides with the respective motor functions assigned to the cerebellum and the cerebrum by Hughlings Jackson.

I believe, on the other hand, in no such distinctive division of function between these two organs existing from the beginning, but that during the process of evolution both of the animal series as a whole and of the separate individuals by which it is now represented, there has been, and is, a gradually more and more complete assumption by the cerebellum of supreme influence over the execution of all automatic and constantly recurring acts. As I have elsewhere pointed out ('Brain as an Organ of Mind,' pp. 568, 555-563) the cerebrum and the cerebellum seem to be intimately associated with one another during all attempts to perform new and unaccustomed movements; but in the production of automatic movement it seems probable that in proportion to their degree of automaticity, the cerebral share in their production becomes less and less, whilst that of the cerebellum becomes more and more preponderant. This gradual handing over of the dominant influence in the production of movements from the cerebrum to the cerebellum, as the movements themselves pass in the development of the race from the voluntary to the automatic category, would seem to be only possible under two conditions—for the actual existence of each of which distinct evidence is forthcoming. It would seem (*a*) to be essential that the cell and fibre mechanisms upon whose existence the ultimate production of the movements more immediately depends should be established in the spinal cord and the medulla oblongata (*loc. cit.* pp. 558-562), which appears to be actually the case, although under the influence of the cerebrum and the cerebellum new combinations, or higher groupings, of these more elementary mechanisms for movements may be brought about. But, if the execution of movements which have become habitual, and still more those which have become actually automatic, is to be gradually handed over in more and more completeness to a maximum of superintendence on the part of the cerebellum and a minimum of superintendence on the part of the cerebrum, it would seem also (*b*) to be essential that there should be a double set of afferent channels, the one set conveying to the cerebrum the sensory incitations to movements, and the other set for conveying to the cerebellum similar incitations. And there is abundant evidence to show that the cerebellum receives through its lower and its upper peduncles afferent impressions of almost all kinds (*loc. cit.* pp. 503-506) which may, in that organ, act as instigators of movement, and yet may not in any way reveal themselves in consciousness. It was the extent of the connections of the cerebellum with different orders of afferent fibres that formerly led to the mistaken notion of Foville and others, that this organ was a kind of 'sensorium commune.' Of course, relations with sensory nuclei or centres are indispensable for a motor centre, whether its position in the hierarchy of such centres be high or low; only, the higher it is, the more numerous are these connections likely to be.

It seems to me very probable, also, that even in voluntary acts the cerebellum plays an all-important part at the instigation and, as it were, under the direction of the cerebrum. I believe it to supply, not the motor energy, for that is yielded by the muscles themselves, but the constant flow of molecular incitations needful, during the continuance of movements for evoking the continuance of contractions in the muscles employed. The cerebrum may incite, continue, or change, this or that form of voluntary movement; but in doing so, I believe it plays upon the cerebellum, and causes this great well-spring of nervous energy to pour out its pent-up molecular movements in an appropriate manner upon the various muscles concerned in the production of the desired movements, just as long as the desire to evoke such movements continues. The comparative sameness of structure throughout the whole cortex of the cerebellum, the extraordinary way in which by foldings of the surface this cortex is multiplied in its extent, and the comparative size of the organ in which all this is brought about, make it not unreasonable to suppose that the cerebellum may be a great storehouse of molecular energy, destined to be used, in different ways, in actually evoking the immense number of muscular contractions which are constantly taking place in our bodies even under conditions of comparative rest, as well as of meeting those enormous drains of nervous energy, needful for the same purpose, during long hours of active exercise or labour.

It was in accordance with these notions that in 1880 (*loc. cit.* p. 509) I ventured to say, "If we are to attempt shortly to sum up its functions, it may be said that the Cerebellum is a supreme Motor Centre for reinforcing and for helping to regulate the qualitative and quantitative distribution of outgoing currents, in Voluntary and Automatic Actions respectively.

New experiments that have been made since the above mentioned date, are far from being incompatible with these views. Up to within a recent period it had been found impossible by experimenters to remove the cerebellum from any of the higher animals and to retain them alive sufficiently long for the immediate irritative effects of the traumatism to subside, so that the mere effects upon the animal of the removal of this organ might be witnessed. Recently, however, this has been achieved by Prof. Luciani, who has embodied the results of his observations in an important memoir published in the 'Revista Sperimentale di Frenatria,' 1884. He succeeded, after complete removal of the cerebellum from a dog, in keeping the animal alive and under observation for eight months. We may here disregard the symptoms produced during the first six weeks, which may have been in part due to irritative and inflammatory processes affecting important parts contiguous to the cut cerebellar peduncles, and merely mention, as a point of extreme interest, that during the last two months of its life, the animal, though living in the midst of the same general influences as before, began to manifest various trophic changes, both local and general—the most important being "a rapidly progressive denutrition, culminating in a peculiar marasmus, or general dystrophy, which suggested a possible causal relation between it and the absence of the cerebellum." What is now of greatest interest to us is the state of the animal during the intervening four or five months; and this is what is said on the subject by a writer in the 'British Medical Journal' (Dec. 6, 1884, p. 1148) in his account of Prof. Luciani's researches—whose memoir I have not yet been able to see:—

"As the process of recovery advances, the symptoms just described fade away, and those of the second period, those dependent upon the actual loss of the organ, come to the front. The so-called incoordination (contractures and pseudo-paralytic debility) disappears, the animal can walk for longer and longer distances without falling. What strikes the observer is the deficient proportion, firmness, and tone of the individual muscular contractions, and their want of fusion; in a word, a kind of 'cerebellar ataxy,' difficult to describe accurately. This disorder of the movements is not, as the 'incoordination' of the first period, sufficiently serious to prevent the animal from effecting its various voluntary acts; and upon closer investigation, resolves itself into a kind (not of paralysis nor paresis, but) of motor 'asthenia.' For instance, if, instead of walking, the dog were made to swim in a pond, then its movements were perfectly normal. Not the least sign of want of equilibration was noticeable; the four limbs acted with ease, the animal being propelled in the water just as it had been noticed to be before the operation, showing a full co-ordination and adaptation of the individual movements necessary to the performance of the action. But, on arriving at the edge of the pond, the dog was unable to get a footing, in spite of its efforts. Professor Luciani lays stress upon the great difference between the normal behaviour of the dog whilst swimming, and its disturbed motility when performing acts requiring a greater development of muscular energy: and concludes that the 'cerebellar ataxy' just described, is merely a manifestation of a kind of motor asthenia, that is to say of the insufficient muscular tone, or influence exercised by the nerve-centres upon the voluntary muscles."

These facts are extremely suggestive and interesting, but taught by experience, derived from other experimental results, we must not be too prone to assume that what has been found to occur in the dog would similarly occur in the human being. It would seem certain that a more complete differentiation of function has been brought about in the great nerve centres in man, than in those of the dog. Consequently we find losses of function temporary in this animal which would have been permanent in the human being. Destruction of the Rolandic region of the cortex, for instance, in the dog produces only a temporary hemiplegia, whilst in man the hemiplegia due to a similar lesion has been shown by the results of disease to be of a permanent character. So also, we might find that destruction of the cerebellum, of an uncomplicated character, might lead in man to more marked and more durable effects than they have been found to produce in the dog.

What sort of explanation can be given of the partial recovery of power in the dog above referred to? Seeing that there is, or has formerly been, a community in functional activity on the part of the cerebrum and the cerebellum in the production of all kinds of movements, it is quite legitimate to suppose that in cases where the cerebellum has been destroyed the cerebrum may be able to take on again that major share in the production of movements, which we assume it to have had during the times when such movements were being learned. We should, in such a case, have to do with just that kind of physiological substitution, for which there was the most reasonable basis of probability, seeing that it would merely necessitate the reopening of old and partially disused nervous channels. Thus, it seems not at all improbable that, in the dog operated upon by Luciani, the effects of the complete removal of the cerebellum may have been distinctly diminished after the lapse of some weeks, by a considerable resumption on the part of the cerebrum of a kind of motor activity which it had previously ceded to the cerebellum. In the event of this being true, the condition of the dog, as observed by Luciani, between the end of the sixth week and the end of the sixth month from the date of the operation, would be far from representing the sum total of effects that might be expected to result, in man, from complete ablation of the cerebellum. In the dog we meet with only a certain residue of functional disability; but there is much reason to believe that after anything like a similar destruction of the cerebellum in man, the permanent disability as regards the execution of movements would have been, and that to a decided extent, both deeper and wider. (See also **Paralyses due to Lesions of the Cerebellum.**)

XXI. TROPHIC CHANGES IN THE JOINTS, NERVE TRUNKS, AND MUSCLES OF PARALYSED LIMBS, IN HEMIPLEGIC PATIENTS.

Trophic lesions are met with only in some cases of hemiplegia, and it will be found that they occur only in some of the cases in which the pyramidal tract has been destroyed in some part of its course, and, therefore, in cases in which a secondary degeneration has been set up in the posterior part of the lateral column on the paralysed side.

We shall look first of all to the facts, and subsequently consider the mode in which they are to be interpreted.

(a).—*Inflammation of joints.* It was originally pointed out by Dr. Scott Alison that a subacute inflammation of some of the joints on the paralysed side of the body—either in arm or leg, but most frequently in the former—occasionally shows itself in from three to six weeks after the onset of a hemiplegia. Rarely, it shows itself a little earlier or later than the period above mentioned. The condition has subsequently been investigated by Brown-Séquard and Charcot. The joints become slightly red, swollen, and painful when moved; the sheaths of the tendons near the joints are also sometimes affected. In cases where patients, so affected have died, Charcot has found all the characteristics of a subacute synovitis in the affected joints.

Frequently, after a time, the joint affection gradually subsides; and in some cases it may, from the first, be so slight as only to reveal itself by trifling pains when the limb is moved.

(b).—*Changes in nerve trunks.* In the same class of cases as those to which I have just referred, tenderness occasionally exists in different parts of the arm or of the leg (but more especially in the former) instead of being limited to the joints; and where this occurs, as Cornil originally pointed out, we find the pain on pressure more especially marked along the course of the principal nerve trunks.

Cornil ascertained that this tenderness, in the very rare cases in which it was met with, was occasioned by a sub-inflammatory hypertrophy of the nerves and of their sheaths. The nerve trunks were found to be notably enlarged and much more vascular than natural, the enlargement being due to an overgrowth of the connective tissue elements entering into their composition.

In cases where unprovoked 'pains' are complained of in the paralysed limbs, they are most frequently traceable to this condition of the nerves, either alone, or to this in combination with the joint affection. But in some rare instances, in which such pains are felt, a tenderness of the limbs generally may be detected—that is, a tenderness not specially limited to the regions of joints or of large nerves.

Such changes in the joints and nerve trunks on the paralysed side of the body generally supervene with, and constitute, part of the early stage of the state known as 'late rigidity;' and in some of such cases we are also apt to meet with the changes in the muscles now about to be described.

(c).—*Trophic Changes in Muscles.* In certain exceptional cases of hemiplegia in which there is evidence to show that some part of the pyramidal tract has been involved, with the consequent setting up of a descending degeneration in the opposite lateral column of the spinal cord, a rapid atrophic process is apt to show itself in the muscles of one or both of the paralysed limbs—but especially in those of the upper extremity.

It is well known that, as a rule, the muscles of the paralysed side, in such cases of hemiplegia as we have just referred to, show only a slow and very gradual process of wasting; and that they retain a normal or almost normal amount of sensitiveness to the Faradic current. But the atrophy to which we are about to refer, is one which sets in some months or years from the date of commencement of the illness; it then makes rapid progress, and is associated not only with a diminution of any pre-existing rigidities in the affected parts but also, it may be, with a rapid diminution in the activity of the affected muscles to the Faradic current—and, in short, a development in them of the 'reaction of degeneration.'

In order to give an account of the cause of this anomaly in the ordinary progress of a hemiplegic patient, something must be said in reference to the relations existing between the nerve fibres constituting the pyramidal tract and the grey matter of the cord.

It is now generally believed that the progressive diminution in bulk of the fasciculus of nerve fibres constituting the pyramidal tract, as it passes from the cervical to the lumbar region in the lateral column of the spinal cord, is due to the fact that as successive levels in the cord are reached, different groups of fibres of this fasciculus terminate their course by entering into organic relations with the great ganglion cells in the anterior cornu—if they do not pass by way of the anterior commissure to some region in the opposite half of the cord.

The only other supposition which has been advanced is that some, at all events, of the pyramidal fibres pass at different levels directly into the anterior spinal roots, and thence direct to the muscles. In the case of such an arrangement existing, there would be an unbroken connection between the giant nerve cells in the excito-motor region of the cerebral cortex and certain muscles by means of these pyramidal fibres. There are, however, strong reasons for believing that no such anatomical dispositions exist. Thus, Flechsig has found that a study of the anatomy of the spinal cord in the newly born child shows that whilst the development of the pyramidal tracts is as yet scarcely outlined, that of the cells of the anterior cornua and of the anterior roots is already in a very advanced state. This would strongly tend to prove that there is no continuity between the fibres of the pyramidal tracts and those of the anterior roots. This again is distinctly shown by the fact originally pointed out by Bouchard, that even the best marked secondary degenerations in the spinal cord do not, of themselves, lead to any alteration in appearance, or atrophy, of the anterior spinal nerve roots.

There is little positive evidence to show that many of the fibres of the lateral pyramidal tract reach the opposite side of the cord by means of the anterior commissure, although there are some facts tending to suggest that in certain rare cases where the decussation has been very imperfect in the bulb, such decussation may be supplemented by another occurring in some part of the dorsal region of the spinal cord.

The view which seems to be altogether the most tenable is that the fibres of the pyramidal tract, at different levels pass inwards and forwards into the grey matter of the anterior cornua, probably after the axis cylinders have broken up into a series of fibrils.

The existence of these communicating fibrils can, according to Brissaud, be made out much more readily in some cases in which there is a process of sclerosis, either primary or secondary, taking place in the pyramidal tract, than in the healthy spinal cord. From the antero-

Cornil ascertained that this tenderness, in the very rare cases in which it was met with, was occasioned by a sub-inflammatory hypertrophy of the nerves and of their sheaths. The nerve trunks were found to be notably enlarged and much more vascular than natural, the enlargement being due to an overgrowth of the connective tissue elements entering into their composition.

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The only other supposition which has been advanced is that some, at all events, of the pyramidal fibres pass at different levels directly into the anterior spinal roots, and thence direct to the muscles. In the case of such an arrangement existing, there would be an unbroken connection between the giant nerve cells in the excito-motor region of the cerebral cortex and certain muscles by means of these pyramidal fibres. There are, however, strong reasons for believing that no such anatomical dispositions exist. Thus, Flechsig has found that a study of the anatomy of the spinal cord in the newly born child shows that whilst the development of the pyramidal tracts is as yet scarcely outlined, that of the cells of the anterior cornua and of the anterior roots is already in a very advanced state. This would strongly tend to prove that there is no continuity between the fibres of the pyramidal tracts and those of the anterior roots. This again is distinctly shown by the fact originally pointed out by Bouchard, that even the best marked secondary degenerations in the spinal cord do not, of themselves, lead to any alteration in appearance, or atrophy, of the anterior spinal nerve roots.

There is little positive evidence to show that many of the fibres of the lateral pyramidal tract reach the opposite side of the cord by means of the anterior commissure, although there are some facts tending to suggest that in certain rare cases where the decussation has been very imperfect in the bulb, such decussation may be supplemented by another occurring in some part of the dorsal region of the spinal cord.

The view which seems to be altogether the most tenable is that the fibres of the pyramidal tract, at different levels pass inwards and forwards into the grey matter of the anterior cornua, probably after the axis cylinders have broken up into a series of fibrils.

The existence of these communicating fibrils can, according to Brissaud, be made out much more readily in some cases in which there is a process of sclerosis, either primary or secondary, taking place in the pyramidal tract, than in the healthy spinal cord. From the antero-internal aspect of the pyramidal tract it is, in these morbid states, easy to see more or less condensed groups of fibres detach themselves and make for the grey substance in which they are lost to view.

In the grey matter itself these fibrils may come into relation with branched processes from the great ganglion cells—this, however, is only a probable hypothesis; all that we know for certain is that there are distinct indications of fibrils passing in the direction indicated between the pyramidal fasciculus and the anterior cornu. In the case of such structural relations existing between the pyramidal fibres and the ganglion cells of the anterior cornu, we should have to regard the pyramidal tract as an aggregation of internuncial fibres, connecting the great cells of the cortex with different groups of cells in the anterior cornu of the spinal cord.

The number of instances in which atrophy has supervened in cases of hemiplegia associated with late rigidity, and has been followed by a thorough examination ot the spinal cord, is extremely limited. There are, however, on record cases that seem to illustrate different modes by which the degeneration in the pyramidal tract may become, as an exceptional event, complicated with an atrophic process in the great ganglion cells of the anterior cornua, and, as a consequence of this, with the well marked muscular atrophy in the muscles of the paralysed limbs with which we are now concerned.

(1). In the first and, indeed, in some of the succeeding cases observed by Charcot he distinctly intimates that the process of sclerosis existing in the pyramidal tract had extended beyond the usual limits in the lateral columns and had invaded the contiguous grey matter of the anterior cornu, giving rise to diminution in bulk of this part as a whole, to atrophy of its ganglion cells, and to overgrowth of its neuroglia ('Localisation of Cerebral and Spinal Diseases,' p. 139). Hallopeau speaks also of having seen similar cases, in which the atrophy of the grey matter had been brought about by an extension of the lateral sclerosis beyond its usual bounds so as to invade the anterior cornu.

(2). Pitres in 1876 observed and carefully examined another of these cases in which, however, the wasting of the nerve cells seems to have been occasioned in an entirely different manner. In this case paralysis had been caused by a hæmorrhage occupying the middle third of the internal capsule on the right side. The muscular atrophy, which had attracted attention during life, affected the left upper extremity almost in its entirety. On the other hand, the muscles of the paralysed lower limb exhibited no notable atrophy. Pitres found, on careful microscopical examination, a descending degeneration on the left side throughout the whole length of the cord; but, in addition, at a limited point of the cervico-brachial enlargement, between the seventh and the ninth pairs, there was in the anterior cornu a complete disappearance of most of the cell groups normally seen in this region (posterior and antero-external groups). The anterior roots also in the corresponding regions of the cervico-brachial enlargement were much greyer than those on the opposite side.

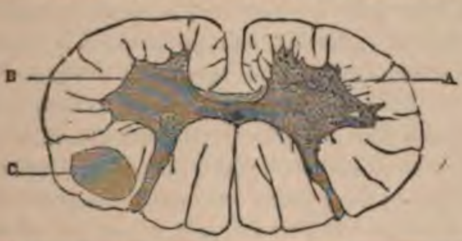

FIG. 27. SECTION OF THE SPINAL CORD BETWEEN THE SEVENTH AND NINTH NERVE ROOTS, IN WHICH ATROPHY OF GANGLION CELLS IN THE ANTERIOR CORNU CO-EXISTED WITH A DESCENDING DEGENERATION IN THE LATERAL COLUMN [after Pitres].

A, anterior cornu on the right side containing the normal number of healthy motor cells. B, left anterior cornu, in which the motor cells have disappeared, except at the most external and at the antero-internal part. C, degeneration of the pyramidal tract in the lateral column.

In this important case it would seem to be perfectly clear that the atrophy of the nerve cells in the anterior cornu was not brought about by a quasi-accidental extension of the sclerosis from the lateral column to the grey matter, after the manner previously noted by Charcot and also by Hallopeau. The degenerated region was separated by a distinct tract of white matter from the grey cornu, so that there was no evidence of any direct extension of the lesion in the lateral column to the grey matter, such as was reported to have been the cause in the previous cases. It seems here to have been due to some more subtle influence, which can only be explained in accordance with the commonly entertained view that the fibres of the pyramidal tract have direct functional relations with the ganglion cells of the anterior cornu, and that the two kinds of elements are connected by means of inter-communicating fibrils. As a rule, as Charcot says—" When there is secondary degeneration of the lateral column, the cell, by reason of its autonomy, generally resists invasion of the morbid process and, if we may use the expression, protects the corresponding anterior root . . . But in certain exceptional cases which are perhaps not so uncommon as is usually believed, under the influence of unknown conditions the cell itself is affected with atrophy, and consecutively the corresponding roots undergo degenerative disintegration."

Whatever may be the precise manner in which it is brought about, when once the atrophic process has been set up in the anterior ganglion cells in any part of the cord, atrophy of the nerve roots in relation with them is bound to follow, and coincidently with this an atrophic process is established in the muscles with which such nerve fibres are in relation. In fact, according to the rapidity with which the process of atrophy is set up in the ganglion cells, we may get an atrophy of muscles in the affected limb which sets in either after the manner of a sub-acute or of a chronic spinal paralysis. At other times, it occurs more after the fashion of a progressive muscular atrophy; and in that case the reaction of the muscles to Faradisation does not disappear during the progress of the atrophy. This process may show itself in arm or leg, but is more frequent in the former; it has not as yet been detected in the trunk muscles. In the upper extremity it is most readily detected in the muscles about the shoulder joint, and in those around the thumb. Its onset or progress is sometimes marked by pains in the affected muscles. If this process should become established in a limb in which some amount of motility had persisted (which, however, is not very likely to happen), the patient's condition would be decidedly aggravated, because as the muscular atrophy progresses the limb becomes more and more flaccid and useless.

It is only in very exceptional cases that such a process of rapid atrophy sets in within a few weeks of the onset of hemiplegia. Still I have recorded one such case in which it showed itself within nine weeks from the date of onset ('Paral. from Brain Disease,' p. 170). It may not declare itself for several years after the limbs have been in a condition of confirmed contracture. In such a case the change in the state of the limbs is most marked; the rigid condition gradually disappears and gives place to one of complete flaccidity, unless it should happen that the fixed position has existed so long as to lead to adaptive changes in the joints of such a nature as not to permit the return of the limb to a condition of complete flaccidity. In a case of this kind, however, the remaining rigidity would be solely due to secondary changes in the joints and not to the condition of the muscles themselves.

XXII. ON CERTAIN OTHER TROPHIC CHANGES IN CONNECTION WITH BRAIN LESIONS.

In addition to the sequential changes to which we have already referred, as taking place in the spinal cord, in the muscles, and in the joints, there are other occasional trophic sequences of brain lesions which we ought to mention, even though nothing very definite can be said, as yet, as to the regional indications afforded by such changes.

(a). *Acute Sloughing* (Decubitus acutus) is a very rare trophic lesion that occasionally manifests itself even within three or four days from the onset of an apoplectic attack by which a hemiplegia commences. This is an acute process of sloughing, which, independently of external irritating influences, occasionally shows itself, not over the mid-sacral region as in spinal disease, but over the centre of the gluteal region on the paralysed side. Charcot says he has seen this occur even when the most minute precautions have been taken in order to prevent it—that is, where the patient has, from the first, been made to lie on the non-paralysed side, and where all contact with urine has been most carefully prevented. At the same time, the liability to sloughing on the paralysed side, under the influence of irritation, is notably increased in these cases, so that such a process is very apt to occur on the heel, on the inner side of the knee, and, in fact, wherever the skin is exposed to slight though continuous pressure.

The acute gluteal slough commences in the form of an erythematous patch. On this, bullæ and hæmorrhagic spots soon appear, and these are followed by a necrosis of the skin which spreads concentrically. The patient may not live long enough for the slough to separate; for this lesion is a complication seldom occurring except in cases about to prove fatal. Nothing definite can at present be said concerning the situation of the cerebral lesions with which this trophic change is most prone to manifest itself. It has, moreover, been known to occur with hæmorrhage, with softening, and also after traumatic injuries of the brain.

(b). *Congestions and Hæmorrhages* are also occasionally produced at the same early stage of the disease, though these, on account of their situation, are only recognizable where the malady terminates fatally. Among the principal of such lesions, we must include congestions and actual hæmorrhages into the lungs; though there may also be minute extravasations beneath some of the serous or mucous membranes. Both Schiff and Brown-Séquard have been able to produce hyperæmic conditions or actual hæmorrhages in similar situations, experimentally in some of the lower animals—by means of traumatic irritation of the pons and middle cerebellar peduncles, and, to a less extent, by irritation of the optic thalami and corpora striata. Brown-Séquard concludes from his experimental observations on animals, and from the fact that these lesions are most readily produced by injuries of the pons Varolii, that they are probably brought about by sudden vaso-motor contractions leading to rupture of capillaries in the sites of hæmorrhage. The principal practical bearing of such experiments, is to be found in the explanation which they afford of the congested and hæmorrhagic condition of the lungs, apt to be met with (in the absence of other determining conditions) in many severe cases of cerebral apoplexy.

(c). *Dropsy of Paralysed Limbs* is met with occasionally in cases of hemiplegia. When present it usually manifests itself in both arm and leg, or in either of them singly, in from three to six days after the onset of hemiplegia. It is mostly a complication of slight significance which tends to abate after a time. There may be conditions in the paralysed limbs which favour the transudation of serum, or it may be that some alteration in the nervous influence retards the rate of absorption. Such a tendency is, however, most apt to manifest itself when some other conditions predisposing to the occurrence of dropsy exist—such as some incipient renal disease, or the existence of an anæmic state of the system.

(d). *Retardation of Rate of Growth in Paralysed Limbs* is a trophic result which often manifests itself when severe brain lesions occur in infancy or early childhood. The cases that have come under my observation have almost all of them been instances of right-sided paralysis ('Paralysis from Brain Disease,' p. 180), and I am disposed to think that the left hemisphere is, in infancy, more prone than the right to be the seat of such lesions as cause this retarded rate of growth. This kind of trophic defect has already been referred to (see p. 49) so that we need say nothing further on the subject here, except that a retarded rate of growth in the finger nails on the paralysed side is also to be observed in adults, as Weir Mitchell has pointed out.

CLINICAL INDICATIONS FAVOURING THE DIAGNOSIS OF DISEASE IN THIS OR THAT ENCEPHALIC REGION.

Under this head we have now to consider the signs and symptoms indicative of lesions in different parts of each cerebral hemisphere, as well as in its peduncle, in the pons Varolii, in different parts of the cerebellum, and lastly in the bulb.

We have already indicated that, in the great majority of cases, a lesion of one hemisphere which involves the motor region reveals itself by paralysis on the opposite side of the body. The exceptions to this rule are of extreme rarity, and as their probable cause has been already referred to (p. 161), it is unnecessary now to say anything further on the subject.

No such cross relation, however, obtains in the cases of paralyses due to lesions of single cranial nerves, where any of these have been implicated by disease or injury in some part of their course between the surface of the brain and the periphery.

In a certain number of cases loss of speech, of the aphasic type, may present itself alone—that is without paralysis of limbs or face. This defect of speech is of a distinctly paralytic nature, and yet owing to the muscles concerned being bilateral we have no means of applying the rule above given for deciding as to which side of the brain is at fault. A combination of clinical and of pathological evidence relating to such cases, and also to the much more common types of disease in which such aphasic defects are associated with a more or less complete hemiplegic condition, has however taught us that in the great majority of cases of this kind the diagnostic indication is safe, that we have to do with disease in the left cerebral hemisphere (p. 119).

In fact, a long series of observations has compelled us to recognize the greatly superior activity of the left hemisphere, as compared with the right, in initiating motor acts subservient to intellectual expression. Just as the left hemisphere has undoubtedly to initiate the muscular acts by which writing is effected in right-handed individuals, so, it would appear that, from this same half of the brain the incitations habitually pass over which are destined to excite the motor acts of speech—even though the muscles concerned are bilaterally disposed and always act in concert on the two sides of the larynx, fauces, tongue and lips (p. 176).

There is reason to believe that this initiatory action of the left hemisphere in relation to speech-movements is connected with a very slight precedence in its development as compared with that of the opposite hemisphere—and that this precedence is itself a more or less remote consequence of an inherited tendency to right-handedness. Accordingly, it has been found in some well-marked cases, that aphasia when occurring in left-handed persons goes with left rather than with right-sided hemiplegia—that is, with lesions in the right hemisphere of the brain.

In regard to the nature of the lesions occurring in different encephalic regions, it is worth while here to mention the fact that so far as lesions of vascular origin are concerned, hæmorrhages are far more frequent than softenings in regions supplied by the vessels of the basal system, viz., in the internal capsules, the corpora striata, the thalami, and the cerebral peduncles ; whilst, on the contrary, in the provinces supplied by the cortical system of vessels, ischæmic softenings are of common occurrence, and hæmorrhages are comparatively rare.

In addition to considering in detail the signs indicative of disease in this or that encephalic region, it will be desirable to consider pretty fully those indicative of paralysis or of irritation of the several Cranial Nerves. In regard to some of them also (and this applies to sensory nerves more especially), we shall have to refer to the intra-encephalic course taken by their fibres on the way to their respective cortical termini.

We now strive to differentiate the effects of lesions in far more numerous encephalic regions than was formerly possible or even thought of, and of late years much more precision has been introduced into the whole subject. Undoubtedly the experimental observations of Hitzig (1870) and of Ferrier (1873) on the cortex of the hemispheres have contributed greatly to bring about this very desirable result. These important experimental observations have added much to our knowledge, though still more of reliable information for us has been derived from the clinico-pathological enquiries which they have stimulated, as well as from other investigations of a developmental order.

It is almost needless to say that previous to this period the problems connected with the localisation of disease in different parts of the encephalon were receiving the earnest attention of many workers; that many valuable results had already been accumulated; and that the gains to our knowledge accruing from the experimental labours above mentioned have been mainly in the direction of an increased ability to discriminate the effects resulting from lesions in one particular part of the cortex, viz., the Rolandic area. Outside this area of the cortex the distinctive effects of lesions are either unknown or are still the subject of much discordant opinion. Some enthusiasts speak as though the doctrine of 'cerebral localisation' were one of quite recent growth, and as if the only attempt at localisation worthy of the name were to be that which concerned itself with surface lesions.

We shall now endeavour to epitomise as briefly as possible the signs and symptoms indicative of disease in the following Encephalic Regions. We adopt the kind of division which seems best calculated to meet the purely practical object that we have in view in this work.

The special regions to which we shall direct our attention will be found to fall pretty accurately into three groups, according as they come within the territory supplied by the Cortical Arterial System, the Basal Arterial System, or those which are exclusively nourished by the Vertebral and Basilar Arteries.

Arterial supply is, therefore, made the basis of our classification; and the consideration of the symptoms produced by lesions in the several departments of each of these groups of territories is prefaced by an account of the mode in which their vessels are distributed.

This part of our subject will, therefore, be treated in the following order:—

I. LESIONS IN PARTS SUPPLIED BY THE CORTICAL SYSTEM OF VESSELS.

(A). Distribution of Vessels Pertaining to the Cortical Arterial System.

CORTEX CEREBRI.
- 1.—Præ-Frontal Region.
- 2.—Occipital Region.
- 3.—Parieto-Temporal Region.
- 4.—Rolandic Region.

CENTRUM OVALE.
CORPUS CALLOSUM.

II. LESIONS IN PARTS SUPPLIED BY THE BASAL ARTERIAL SYSTEM.

(B). Distribution of Vessels Pertaining to the Basal Arterial System.

INTERNAL CAPSULE.

1.—Direct efferent fibres.
2.—Direct afferent fibres.

BASAL GANGLIA.

1.—Caudate Nucleus.
2.—Lenticular Nucleus.
3.—Optic Thalamus.
Hæmorrhage into the Lateral Ventricles.

CORPORA QUADRIGEMINA.

CRUS CEREBRI.

1.—Lower and Inner Portion.
2.—Upper and Outer Portion.

III.—LESIONS IN PARTS SUPPLIED BY THE VERTEBRAL AND BASILAR ARTERIES.

(C). Distribution of the Vertebral and Basilar Arteries.

PONS VAROLII.

1.—Central Region.
2.—Lower half of one Lateral Region.
3.—Upper half of one Lateral Region.

CEREBELLUM.

1.—Lateral Lobes.
2.—Median Lobe.
3.—Middle Peduncles.

BULB (MEDULLA OBLONGATA).

CRANIAL NERVES.

A. Olfactory Nerve.
B. Optic Nerve.
C. Third Nerve.
  Fourth Nerve.
  Sixth Nerve.
D. Fifth Nerve.
E. Facial Nerve.
F. Auditory Nerve.
G. Pneumogastric Nerve.
H. Glossopharyngeal Nerve.
I. Spinal Accessory Nerve.
J. Hypoglossal Nerve.

Nothing like finality or great precision is to be expected in dealing with this part of our subject. Our knowledge, in regard to some parts of it, is still involved in much obscurity; but in this, as in all other difficult enquiries, it is eminently useful clearly to recognize the real boundaries of our knowledge. We thus pave the way for further observation, which may enable us to fill in this or that empty space in our chart, or to render more precise this or that information previously given.

In endeavouring to systematise what is known upon this subject we must necessarily proceed in a simple and comparatively artificial manner. The encephalon must be mapped out into a number of more or less arbitrary regions, such as we have tabulated above, and we must consider separately the effects of a single lesion in each one of these regions. In actual practice the problems presented are often very complex; we may have to do, for instance, with simultaneous lesions of more than one part, or with lesions situated on the confines between two of the artificial encephalic departments, the symptomatology of which is considered separately. In all such complications we must use our knowledge of the more simple cases to try to unravel the meaning of the more complex symptomatology. It may be stated, however, as a general rule, that when two or more lesions coexist in the encephalon, anything like a positive regional diagnosis often becomes more or less hopeless.

In dealing with this subject of regional diagnosis, two courses were open, as regards the order in which the different regions were to be considered; we might either begin at the cortex and gradually proceed downwards to the bulb, or we might begin with the bulb and follow with higher and higher regions up to the cerebral cortex. The former course has been deemed to be the most expedient.

It has been thought necessary to give a more special consideration to two encephalic regions, viz., to the Cerebellum, and to the Bulb. And looking to the extreme importance of the different combinations of paralyses of Cranial Nerves often met with as part of the symptoms produced by intra-cranial lesions, as well as to the intrinsic importance of these paralyses and of the settlement of the question which must always present itself in regard to any of them, as to whether such paralyses are due to central or to peripheric lesions, it has been thought advisable to enter fully into this part of the subject.

In dealing with lesions of the Bulb as well as with those of the Cranial Nerves, the questions concerning pathological diagnosis have been considered, as well as those relating to regional diagnosis.

The section concerning paralyses of Cranial Nerves, though it stands last in the order of subjects relating to paralyses of encephalic origin, is one to which frequent reference will have to be made, because, in the case of intra-cranial lesions occupying the most varied situations, the functions of one or more of the cranial nerves is apt to be interfered with. When incidental references have to be made to such paralyses, no descriptions of the corresponding signs and symptoms will be given; because, if exact information be required by the student or practitioner, he must seek it under the proper head in the special part of the work above referred to.

Where points of importance in reference to the blood-supply of the several encephalic regions deserve to be borne in mind, these will be mentioned before calling attention to the signs indicative of lesions in such regions. Many of the facts concerning blood-supply will be found to have significant bearings upon the problems of pathological as well as upon those of regional diagnosis. We call attention to them therefore between the consideration of these two problems.

I. LESIONS IN PARTS SUPPLIED BY THE CORTICAL ARTERIAL SYSTEM.

DISTRIBUTION OF VESSELS PERTAINING TO THE CORTICAL ARTERIAL SYSTEM.

Some few details have already been given concerning the precise mode in which the cortical grey matter is supplied with blood, and what has been there said (p. 14) applies to all three pairs of vessels belonging to the cortical system.

We will now give an abstract of Duret's account of the mode of distribution and principal branches of each of these vessels.

1. The Middle Cerebral or Sylvian Arteries. Outside the limits of the basal circle (bounded by the dotted line in Fig. 1) each middle cerebral enters the fissure of Sylvius and soon passes over the floor of the island of Reil. About this situation it commonly divides into five branches, each of which has received a specific name. The distribution of these branches is shown in Fig. 28 in a diagrammatic manner.

The precise distribution of the middle cerebral to different convolutions is however accurately shown in Fig. 29 in which Duret, making use of the outline figures of the convolutions given by Ecker, maps out thereon the outlines of the several arterial territories and departments.

This and other figures of the same kind will serve a double purpose. They will show the distribution of the cortical system of vessels according to Duret; and they will also serve the purpose originally designed by Ecker, viz., of giving faithful outline sketches, showing the situation and relative arrangement of the several Fissures and Convolutions of the Cerebral Hemispheres.

It will be seen that the branches of the middle cerebral are distributed over a very large proportion of the outer surface of the cerebral hemispheres; that is, over the whole of the parietal region, the upper half of the temporal lobe, and the posterior and inferior part of the frontal lobe.

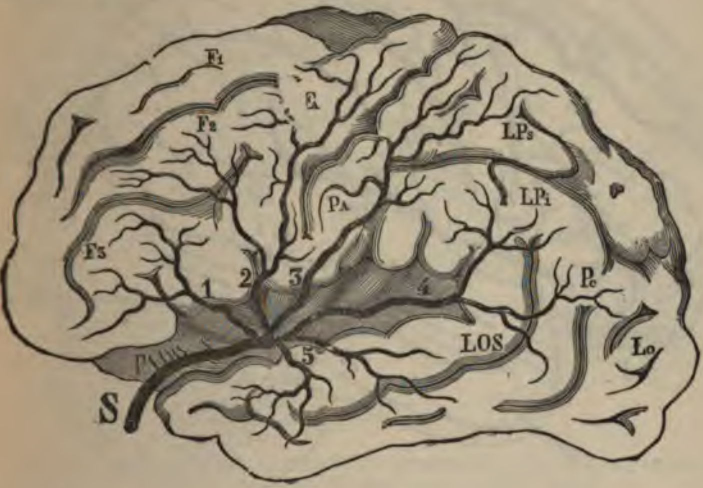

FIG. 28.—DISTRIBUTION OF THE MIDDLE CEREBRAL ARTERY [Semi-diagrammatic view, after Charcot].

S, trunk of the Sylvian entering the fissure of Sylvius, with its branches radiating between the convolutions of the island of Reil; P, perforating branches of the basal system proceeding to the central grey nuclei; 1, external and inferior frontal artery (artery of Broca's convolution); 2, ascending frontal artery; 3, ascending parietal artery; 4, parieto-sphenoidal artery; 5, sphenoidal artery.

F_1, F_2, F_3, first, second, and third frontal convolutions; Fa, ascending frontal convolutions; Pa, ascending parietal convolution; LPs, superior parietal lobule; LPi, inferior parietal lobule; Pc, angular gyrus; Lo, occipital lobe.

2. The Anterior Cerebral Arteries. These vessels are distributed, on each side, to the inner part of the orbital surface of the frontal lobe, to the upper and anterior part of the frontal region, and to the inner surface of the hemisphere as far back as the internal perpendicular fissure.

These arteries, in fact, supply three principal branches which are distributed thus in the regions above mentioned. 1. The anterior and internal frontal arteries supply the two inferior frontal convolutions (Fig. 31). 2. The middle and internal frontal arteries, larger and more important, are often distributed to the convolution of the corpus callosum, to the corpus callosum itself (Fig. 30), to the first frontal convolution (inner and outer surfaces), to the paracentral lobule, and over the convex surface of the hemisphere to the first and second frontal convolutions as well as to the upper extremity of the ascending frontal convolution (Fig. 32). 3. The posterior and internal frontal arteries of each side are distributed only over the quadrate lobe (Fig. 30).

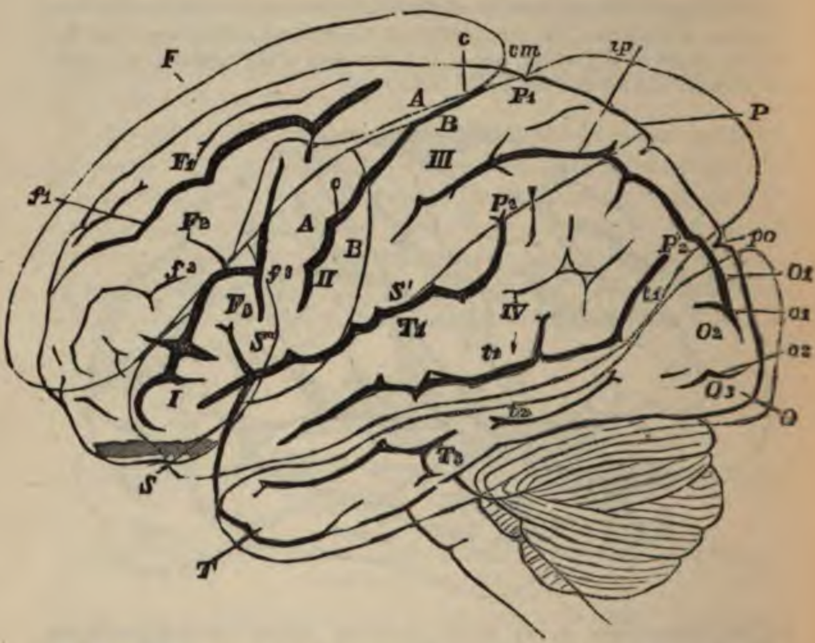

FIG. 29.—OUTER SURFACE OF LEFT HEMISPHERE [after Ecker], SHOWING THE DISTRIBUTION OF CORTICAL VESSELS [after Duret].

F, frontal lobe; P, parietal lobe; O, occipital lobe; T, temporo-sphenoidal lobe.

S, Fissure of Sylvius; S′, horizontal, and S″, ascending ramus of the same. *c*, fissure of Rolando.

A, ascending frontal and B, ascending parietal convolutions; F_1, superior, F_2, middle, and F_3, inferior frontal convolutions; f_1, superior, and f_2, inferior frontal sulcus; f_3, præcentral sulcus.

P_1, superior parietal lobule; P_2, inferior parietal lobule; *ip*, intra parietal sulcus. *cm*, termination of calloso-marginal fissure. *po*, parieto-occipital fissure.

O_1, upper, O_2, middle, and O_3, inferior occipital convolutions. o_1, transverse occipital fissure. o_2, inferior longitudinal occipital fissure.

T_1, first; T_2, second; T_3, third temporal convolutions. t_1, first; and t_2, second temporal fissures.

Distribution of Vessels.

The region bounded by the line (---------) represents the territory over which branches of the Anterior Cerebral Artery are distributed.

The anterior regions bounded by the lines (— - — - — - —) represent the territories over which branches of the Middle Cerebral Artery are distributed.

I. Is the region of the *External and Inferior Frontal Artery.*
II. „ „ *Anterior Parietal Artery.*
III. „ „ *Posterior Parietal Artery.*
IV. „ „ *Parieto-Sphenoidal Artery*, and of the *Sphenoidal Artery.*

The posterior and inferior region bounded by the line (— - - - — - —) represents the territory over which branches of the Posterior Cerebral Artery are distributed.

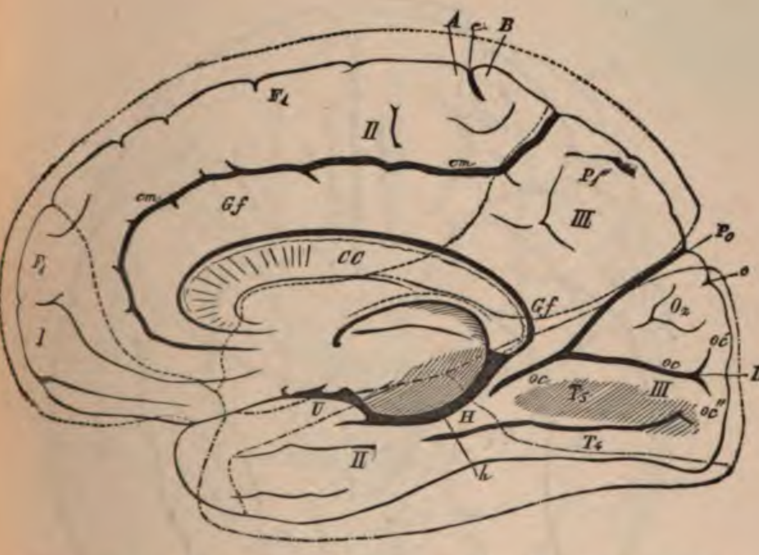

Fig. 30.—Inner Surface of the Right Hemisphere [after Ecker], showing Distribution of Cortical Vessels [after Duret].

CC, section of corpus callosum. A, upper extremity of ascending frontal, and B, upper extremity of ascending parietal convolution. G*f*, convolution of the corpus callosum (gyrus fornicatus). F_1, *cm*, marginal convolution. H, hippocampal convolution. U, uncinate convolution. D, descending convolution (gyrus descendens). P''_1, præcuneus (or quadrate lobe). Oz, cuneus (or occipital lobule). *po*, internal perpendicular (or parieto-occipital) fissure. *oc*, calcarine fissure; *oc'* superior, and *oc''* inferior ramus of the same. H, hippocampal fissure. *cm*, calloso-marginal fissure. *c*, terminal portion of fissure of Rolando (sulcus centralis). *o*, transverse occipital sulcus.

T_4, lateral occipito-temporal convolution (fusiform lobule).

T_5, middle occipito-temporal convolution (lingual lobule).

Distribution of Vessels.

The regions represented by the line (---------) represent the territories over which the branches of the Anterior Cerebral Artery are distributed.

I. Is the territory of the *Anterior and Internal Frontal Artery.*
II. " " *Middle and Internal* " "
III. " " *Posterior and Internal* " "

The regions bounded by the line (- — - — - — -) represent the territories over which the branches of the Posterior Cerebral Artery are distributed.

II. Is the territory of the Posterior Temporal Artery.
III. " " Occipital Artery.

3. The Posterior Cerebral Arteries. Both softenings and hæmorrhages are much more frequent in the territory of this artery than they are in that of the anterior cerebral; in these respects it is much more closely related to the middle cerebral artery.

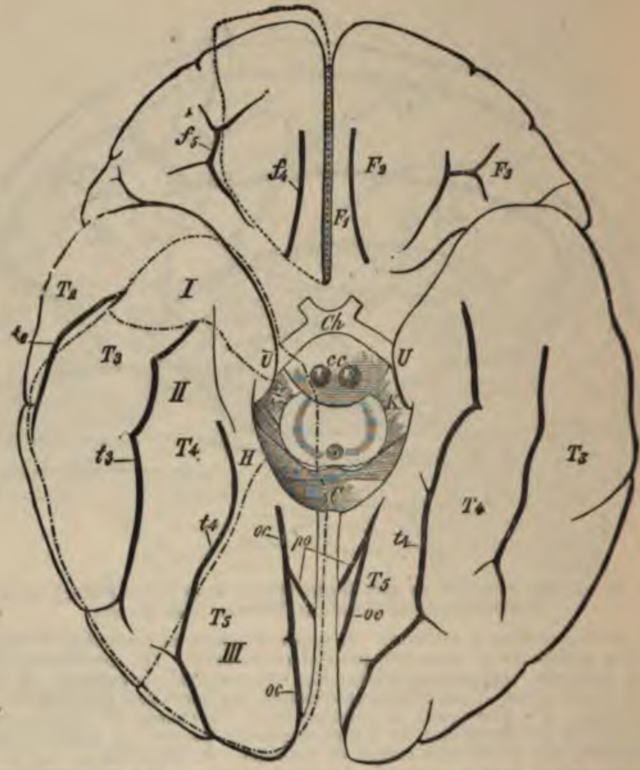

FIG. 31. UNDER SURFACE OF THE CEREBRUM [after Ecker] SHOWING DISTRIBUTION OF CORTICAL VESSELS [after Duret].

F_1, straight convolution (gyrus rectus). F_2, middle frontal convolution. F, inferior frontal convolution. f_4, olfactory sulcus. f_5, orbital sulcus.

T_2, second or middle temporal convolution. T_3, third or inferior ditto. T_4, lateral occipito-temporal convolution (fusiform lobule). T_5, middle occipito-temporal convolution (lingual lobule). t_4, inferior occipito-temporal fissure. t_3, inferior temporal fissure. t_2, middle temporal fissure. *po*, parieto-occipital fissure. *oc*, calcarine fissure.

H, hippocampal convolution. U, uncinate convolution. Ch, optic chiasma. *cc*, corpora albicantia. KK, crura cerebri. C, corpus callosum.

Distribution of Vessels.

The regions bounded by the line (- - - - - - - - - -) represent the territory over which the *Anterior and Internal Frontal* branch of the **Anterior Cerebral Artery** is distributed.

The regions bounded by the line (- — - — - — - — -) represent the territories over which the branches of the **Posterior Cerebral Artery** are distributed.

I. Is the region of the *Anterior Temporal Artery*.
II. " " *Posterior Temporal Artery*.
III. " " *Occipital Artery*.

This artery divides into three secondary branches (Fig. 31). (1). The **anterior temporal artery** supplies the anterior part (crochet) of the uncinate

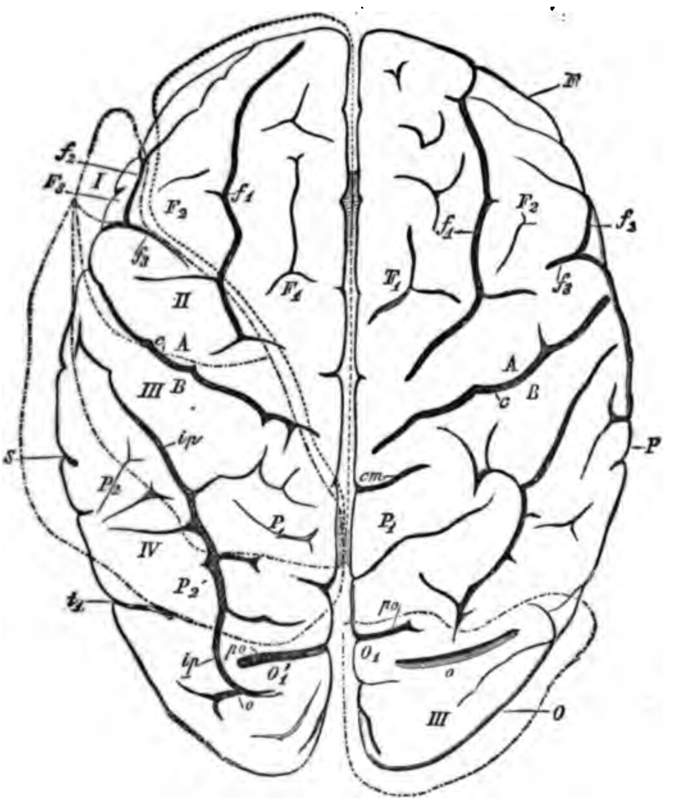

FIG. 32. UPPER SURFACE OF CEREBRUM [after Ecker] SHOWING DISTRIBUTION OF CORTICAL VESSELS [after Duret].

F, frontal lobe. P, parietal lobe. O, occipital lobe. S, end of horizontal ramus of fissure of Sylvius. c_1, fissure of Rolando. A, ascending frontal convolution. B, ascending parietal convolution. F_1, F_2, F_3, superior, middle and inferior frontal convolutions. f_1, f_2, superior and inferior frontal sulci. f, vertical or transverse frontal fissure (sulcus præcentralis). P_1, superior parietal lobule. P_2, inferior parietal lobule (supra marginal convolution). P'_2, angular convolution. *ip*, inter parietal fissure. *cm*, calloso-marginal fissure. *po*, *po*, parieto-occipital fissure. t_1, superior temporal fissure. o_1, first occipital convolution. *o*, transverse occipital fissure.

Distribution of Vessels.

The region bounded by the line (- - - - - - - - - -) represents the territory over which the Anterior Cerebral Artery is distributed.

The regions on the left side of the figure bounded by the line (—— - —— - —— - ——) represents the territory over which the branches of the Sylvian Artery are distributed.

I. *External and Inferior Frontal Artery.*
II. Is the region of the *Anterior Parietal Artery.*
III. „ „ *Posterior Parietal Artery.*
IV. „ „ *Parieto-sphenoidal Artery.*

The region on the right side of the figure bounded by the line (— - — - — - — -) represents the territory over which the Posterior Cerebral Artery is distributed.

convolution, and the under part of the tip of the temporal lobe. (2). The posterior temporal artery supplies the lower part of the temporal lobe, viz., the inferior temporal convolution and the fusiform lobule. (3). The occipital artery supplies the lingual lobule, the cuneus, and the whole of the occipital lobe itself. This last is the more important branch; it may be regarded as the continuation of the main trunk of the posterior cerebral. It turns round to the inner side of the hemisphere, in a position corresponding with the internal parieto-occipital fissure, whence its ultimate branches are distributed over the occipital lobe. It also gives off some important, because comparatively large, branches in the calcarine fissure, which almost immediately penetrate into the adjacent white substance.

The rupture of one or other of these latter twigs occasionally gives rise to larger hæmorrhages than are to be met with anywhere in the midst of the white substance of the hemisphere except in the neighbourhood of the corpus striatum.

Over the whole surface of the hemispheres, as a rule, the vessels do not penetrate till they have been reduced in size to such an extent as to rank almost with capillaries. Hence the paucity of other than miliary hæmorrhages in the cortex and immediately subjacent white matter. In these respects, therefore, the arteries of the calcarine fissure constitute notable exceptions to the rule which obtains elsewhere.

PARALYSES DUE TO LESIONS OF THE CORTEX CEREBRI.

It is desirable to divide the cortex of the Cerebral Hemisphere into certain artificial regions, in order more conveniently to refer to the effects of lesions in different parts of it.

What is now known as the Rolandic region stands out prominently as the area of the cortex in which the occurrence of lesions leads to paralyses of different kinds. It will be found more convenient for our present purpose, however, if we deal with the effects of lesions in this region last, so as to be able to follow it up by the consideration of the effects of lesions of the internal capsule—a part traversed by the efferent fibres from this Rolandic region of the cortex.

Reference will be made, therefore, in succession to the effects of lesions in the following areas of the cortex:—

1. The Præ-Frontal Region.
2. The Occipital Region.
3. The Parieto-temporal Region.
4. The Rolandic Region.

1. Signs and Symptoms of Lesions in the Præ-Frontal Region.— Although the frontal lobe is usually considered to include the three tiers of frontal convolutions together with the ascending frontal convolution, the orbital convolutions and those covering the internal aspect of the corresponding region, yet it is necessary, as Ferrier points out, for physiological and pathological purposes to subdivide it, and to term that part which, in its relation to the skull, is roughly bounded by the coronal suture (see Fig. 33), the præ-frontal region. The remaining portion of the convolutions of the frontal lobe belong, indeed, to the Rolandic region.

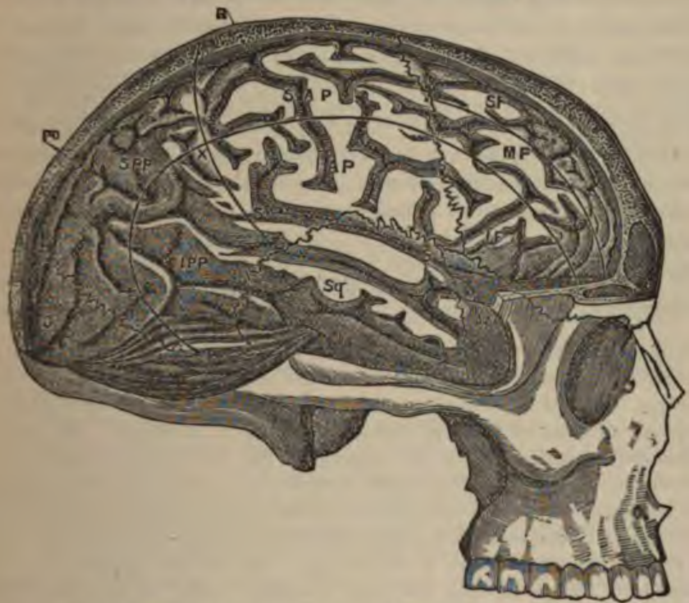

Fig 33. Relations of the Skull and Brain [after Turner].

Ferrier found that electrical irritation of the præ-frontal region in the monkey caused no motor reaction ; and that destruction of these same parts caused no paralysis either of motion or of sensation. Numerous records of injury and disease implicating this region of the brain in the human subject show also, in a most conclusive manner, an absence of any definite impairment of sensibility or motility.

After referring to many of the recorded cases of injury involving this region of the brain, Ferrier ('The Localisation of Cerebral Disease,' 1878, p. 33) says :— "I might multiply instances all demonstrating the same fact, that sudden and extensive lacerations may be made in the præ-frontal region, and large portions of the brain substance may be lost, without causing impairment either of sensation or of motion ; and, indeed, without very evident disturbance of any kind, bodily or mental, especially if the lesion be unilateral." Again, after referring to a number of cases of disease of this region of the brain (principally cases of softening or of abscess) he says:—'In all these cases there was an entire absence of sensory or motor paralysis; and in many there was nothing recorded or nothing calling for special attention as regards the mental condition. In some of them, however, and in one or two others to be referred to, the psychological condition seems to have attracted notice."

In regard to alterations in the mental condition, these may be either non-appreciable or slight in cases of injury of, or disease in, this brain region. Any such symptoms have been very frequently absent where there has been damage to the præ-frontal region of the brain only on one side; whilst, on the other hand, alterations in the mental condition have been not unfrequently noted when these regions have been simultaneously affected in both hemispheres. It has often been difficult precisely to define the nature of the change which has been brought about; but a dull apathetic condition seems to have been most frequently noticed, together with irritability, vaccillation, a diminished power of attention, and a lowering of the moral nature.

Only some of such mental changes could, of course, be detected, even in the highest of the lower animals, in cases where changes of this type had really occurred. Still, it is interesting here to note what was observed by Ferrier as a result of removal of both the præ-frontal lobes in monkeys. He says:—"Removal or destruction by the cautery of the antero-frontal lobes is not followed by any definite physiological results. . . . . And yet, notwithstanding this apparent absence of physiological symptoms, I could perceive a very decided alteration in the animals' character and behaviour, though it is difficult to state in precise terms the nature of the change. The animals operated on were selected on account of their intelligent character. After the operation though they might seem to one who had not compared their present with their past fairly up to the average of monkey intelligence, they had undergone a considerable physiological alteration. Instead of, as before, being actively interested in their surroundings and curiously prying into all that came within the field of their observation, they remained apathetic or dull, or dozed off to sleep, responding only to the sensations or impressions of the moment or varying their listlessness with restless and purposeless wanderings to and fro."

Similar experimental observations have since been made by Horsley and Schäfer. They also found no alterations in sensibility or motility as a result of the operation in monkeys. When one præ-frontal lobe only was excised they noted no changes whatever in the animals' habits and disposition; but where both of these lobes were removed they did, at first, find certain alterations in the disposition and habits of the animals (somewhat similar to those above indicated), though they are of opinion that after the lapse of from three to four weeks the animals showed no appreciable change in manners or disposition from what they had exhibited before the operation.

The case that presents the nearest approach in man to a condition of brain parallel to that which was produced in the monkey by operative interference, is one recorded by Baraduc where there was atrophy of the frontal convolutions in *both* hemispheres, which was not congenital but had supervened during adult life. Ferrier gives the following abstract of this case :—" The patient was an inmate of the Hospice de Ménages for six weeks. His muscular powers and sensation were unimpaired. He was, however, in a state of complete dementia, marching about restlessly the whole day, picking up what came in his way, mute, and quite oblivious of all the wants of nature, and requiring to be tended like a child. The lesion in this case was purely cortical, atrophic, and dependent on partial obliteration of the arterial supply. The lesion occupied the two anterior lobes, that is to say, the first, second, and third frontal convolutions, and also the internal aspect of the frontal lobes. The ascending frontal, ascending parietal, and paracentral convolutions were intact. The rest of the brain was normal except in the region of the inferior parietal lobule of the right hemisphere." It is worthy of note that here the mental defects were permanent rather than temporary and were altogether more marked than those which have been met with even temporarily in monkeys. It should be remembered, however, that in this patient we had to do with disease rather than with injury, and that although there were no macroscopic changes in other parts of the brain, there might nevertheless have been microscopic changes similar to those met with in dementia—in short, that this might have been an ordinary demented patient who presented, over and above what is usual, certain marked macroscopic changes in the frontal lobes.

A similar objection obtains in regard to cases of idiotcy, in which there may have been marked arrest of development or atrophy of the frontal lobes. This condition may coexist with a state of idiotcy dating from birth, but then it is almost always associated with an abortive development (less marked) of other parts of the brain. The same thing may be said concerning general paralysis of the insane in its early stages; it may be quite true that at this period morbid changes are often most obvious in the frontal regions of the hemispheres (Crichton Browne), and also that amongst the earlier symptoms we meet with "general restlessness and unsteadiness of mind, with impairment of attention, alternating with apathy and drowsiness"—and yet these symptoms cannot with any certainty be referred in the main to the lesions in the frontal lobes, because it is probable that less obvious changes may, even in the early stages of the disease, exist in many other parts of the brain.

From what has been said it will be obvious that there are no definite signs by which we can, with any degree of certainty, come to a positive diagnosis that disease exists in either præ-frontal region of the brain. Still, in some cases we may be guided to such a diagnosis by other considerations. The sites of external injury may lead us to infer, in cases of this type, the existence of traumatisms affecting one or both frontal lobes. Again, the existence of disease in the nasal fossæ, or of tumours in the orbit, may, in certain cases, make it probable that the frontal region of the brain has become involved, and this probability may be strengthened, in the latter class of cases more especially, by the existence of impairment or loss of smell on one or both sides. But small or even moderate-sized lesions beginning in the frontal lobes are likely to run either a latent course, or, at least, to be unattended by symptoms which have any localising value.

2. Signs and Symptoms of Lesions in the Occipital Region. We speak of the cortex of the occipital region and of the occipital lobe, next after the frontal cortex and frontal lobe, because lesions in this part of the brain are apt also to run a latent course, or to be devoid of localising symptoms, that is, so long as they are strictly limited to the occipital lobes themselves.

Cases of softening, abscess, tumour, hæmorrhage and traumatism limited to one occipital lobe have been recorded in which there was no appreciable impairment of sensation or motility, and in which, at most, there was some amount of mental obtuseness or hebetude.

Some bilateral lesions in the occipital lobes have also been recorded. In one observed by Sestié there was an abscess in each occipital lobe without any objective symptoms. Although this patient's memory was somewhat defective, there was no other very appreciable alteration in his mental condition. Again, Leger has recorded a case of tumour invading both occipital lobes, in which beyond general mental obtuseness and headache, there were no objective symptoms; sight was not impaired.

In reference to the experimental investigation of this subject with monkeys, Ferrier writes as follows :—"I have found as the result of numerous experiments, that electrical irritation may be applied to the occipital lobes without producing any objectively discoverable reaction. Neither does destruction or complete removal of these lobes singly, or on both sides, cause any appreciable sensory or motor disturbance. Animals so mutilated continue to see, hear, touch, taste, and smell, and retain all their powers of voluntary motion. The results of destruction like those of irritation are, therefore, mainly negative, and do not succeed in throwing very clear light on the functions of these regions. In one or two instances I should mention that there seemed to be some affection of vision ; but in these cases I found that the lesion had extended beyond the occipital lobes, into the angular gyri ; while in those in which the lesion did not extend beyond the occipital lobes, no such symptom occurred." The complex relations of the occipital lobes to vision in cases where they have been damaged alone or in concert with the angular gyrus on one or both sides, as deduced by Ferrier from later experiments, have been elsewhere referred to (p. 145).

From his earlier experiments Ferrier thought that lesions of the occipital lobes (when bilateral) led to a special impairment of the appetite for food. He attempted to explain this on the supposition that "the occipital lobes are specially related to the visceral sensibilities, and are the anatomical substrata of the correlated feelings which form a large portion of our personality and subjectivity" ('Localisation,' p. 117).

The later experiments of Horsley and Schäfer, with monkeys, have tended fully to confirm Ferrier's conclusion that "lesions of the occipital lobes, whether unilateral or bilateral, cannot be regarded as the direct cause either of motor or special sensory affections." They have not noticed any special relation, however, between loss of appetite and excision of these lobes. Monkeys, from whom both occipital lobes had been removed, in their experience, showed no appreciable change in habits or disposition—so that there was no reason for inferring that they had undergone any definite alteration in regard to their mental powers or aptitudes.

Notwithstanding this evidence derived from experimental observations with monkeys, and the other class of facts above referred to, as to the frequent latency of lesions in the occipital lobes, the opinion is held by some physicians that lesions in this region of the brain are more prone than those of an equal size in other regions to be associated with distinct mental defects. Such a view has been expressed by Rosenthal, Hughlings Jackson, and the writer (p. 94). It seems difficult to harmonize the conflicting views and evidence bearing upon this question, and yet a little consideration may suffice to show that the discordance is not so great as it seems.

In this direction, it seems to me that three sets of considerations are worthy of being borne in mind.

1°.—The apparent absence of mental defects in animals from whom the occipital lobes have been removed may be due to the great difficulty in recognizing, or feeling certain about, the existence of any such defect in a dumb animal. So many of its actions belong to the primary or secondary automatic character, that it may be possible for a monkey to seem to be in its ordinary condition, when it would be quite unable, in case of need, to exercise that degree of intelligence which might have been previously possible. The fact that mental defect sometimes seems more apparent in monkeys from whom the præ-frontal lobes have been removed, than in others from whom the occipital lobes have been excised, may not by any means show that the præ-frontal lobes have more to do with higher mental operations than the occipital lobes. In the first place it would be probably wrong to suppose that these great parts of the brain act separately; it is rather a question of more or less, that is of the relative importance of different regions of the brain in this or that kind of functional activity. Thus, if we suppose that the præ-frontal lobes are all important in regard to the manifestation of what is known as 'attention,' and also that they are intimately concerned with emotional states, it does not require much reflection to show us that an animal from which the præ-frontal lobes have been removed may be dull and apparently careless of what is passing around it, when the incitements and first conditions essential for an alert observant attitude are wanting. Such an animal may easily seem to have undergone some very distinct mental alteration.

If we bear in mind the second set of considerations, next to be referred to, it will be found that the suppositions above mentioned are also applicable to the explanation of the effects of disease in the human subject—with the, at times, seemingly greater implication of mind where disease exists in both frontal lobes than when it exists in both occipital lobes.

2°.—In cases of cerebral disease some kinds of mental defect are comparatively easy to recognize, others are extremely difficult. To the former category belong the various degrees of unconsciousness or of delirium; also a dull, apathetic condition (which may result from bilateral disease of the præ-frontal lobes); also certain defects in sensory or perceptive areæ of the cortex (especially those pertaining to hearing or sight in the left hemisphere), making it difficult for the patient to understand spoken or printed words, and perhaps also to express himself correctly. But the kind of defect which is extremely difficult to recognize in a patient suffering from a brain injury or lesion is, for instance, an inability to perform those higher efforts of an intellectual order of which the individual may be capable (the nature of which will, of course, vary widely with education and natural endowment). The reason for this is perfectly clear. Persons who are sick and ill from whatsoever cause, but especially if the cause be an intra-cranial lesion, are not accustomed or usually called upon to exercise their intellect to any appreciable extent—their mental life is carried on in the lower spheres of perception and simple motor response—hence, the existence of any intellectual defect or defect in power of constructive imagination either is not or cannot be detected. This the writer is inclined to think is one of the principal causes why lesions limited to the occipital lobes in man may appear in many cases to be latent, or why, at all events, they cause no easily appreciable mental defect.

3°.—Although what has been said in the previous paragraphs may afford some explanation of the reported absence of mental change in animals and also in man after damage to even both occipital lobes, it does nothing to reconcile the discrepancies between these observations and those other clinical facts upon which Rosenthal, Hughlings Jackson and the writer have been led to form the opinion that lesions in the occipital lobes are especially prone to be associated with mental defects. I am disposed to think that this discrepancy may be in part accounted for by the fact that some observers search more carefully than others for such defects; and in part from the fact that in cases in which lesions in the occipital lobes have been associated with readily detectable mental defects, there may have been an extension of the lesion into the contiguous perceptive centres about the posterior extremity of the Sylvian fissure—the conjoint affection of which sense centres would have the effect not only of intensifying intellectual defects, but of adding to them dulness of apprehension in connection with audition or vision, or defects in intellectual expression such as could not escape observation (see p. 120).

Fresh observations are obviously needed in order to throw further light upon these different points of view—and it is new clinico-pathological facts which are especially required, since experiments upon animals not having the speech-endowments of man, can give us but little information on many of these points. A good illustration of the differences that would result, in the monkey and in the man respectively, from one particular lesion may here be referred to with advantage, owing to the light which it throws upon the difficult subject now under discussion. Thus in a recent paper ('Proceed. of Roy. Soc.,' Jan. 24, 1884) Ferrier and Yeo say :—"Deep incisions may be made in both occipital lobes at the same time, or the greater portion of one or both occipital lobes at the same time may be removed without any appreciable impairment of vision." This is quite in accordance with the conclusion previously arrived at by Ferrier alone, that no sensory defect followed the removal of both occipital lobes. But Ferrier and Yeo also say this :—"The only lesion which causes complete and permanent loss of vision in both eyes is total destruction of the occipital lobes and angular gyri on both sides. . . . Destruction of the occipital lobe and angular gyrus on one side causes temporary amblyopia of the opposite eye and homonymous hemianopia of both eyes, towards the side opposite the lesion." These experiments illustrate the different effects produced by a slight extension of the lesion beyond the occipital region ; and though, taking the case of the unilateral lesion, the additional defect in the case of the monkey was only an impairment of the sense of sight, yet it is perfectly certain that with such a lesion causing destruction of the angular gyrus and of the occipital lobe on the left side in a human being, we should have had also (p. 124) distinct evidence of mental defect and of defect in the patient's power of intellectual expression—since lesion in the left angular gyrus alone may produce what is now commonly known as 'word-blindness.'*

It is held by Munk that removal of, or damage to, both occipital lobes gives rise to an intellectual visual defect, in which, whilst the animal may see and avoid external objects coming in its way, it may do so without apprehending their nature—it does not, for instance, recognize food as such. To some extent this accords with Ferrier's experience, although he puts a different interpretation upon the fact. Goltz has also recognized this kind of visual defect which Munk speaks of as psychical blindness (Seelenblindheit). Whether this species of defect exists or not with unilateral lesions of the occipital lobes must be left for future observation to settle. We may, however, observe here concerning this disability, that it is a lower or more fundamental defect of the same order as ‘word-blindness’; and many considerations make it probable that such a defect would be met with under the conditions above specified—and, according to Hughlings Jackson, especially where the lesion exists in the occipital lobe of the right side.

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* Concerning the possible production of double homonymous hemianopia as a result of lesions of the occipital lobe, see p. 144.

It may be well to note, in this place, that Broadbent has recorded a case of embolism of the right posterior cerebral artery (‘Trans. of Clin. Soc.,’ 1876, p. 61), in which, after death, softening of the outer and lower parts of the right occipital lobe was met with—“the softening extended from the descending cornu of the lateral ventricle to the tip of the [occipital] lobe, and involved the tail of the corpus striatum and the fibres of the thalamus passing to the occipital lobe.” Concerning this patient Dr. Broadbent says:—“Before concluding, I would again call attention to the interesting psychological fact that when the intelligence was so far restored that no deficiency of comprehension, or memory, or reasoning powers was evident—the patient was so indifferent to common decency as to throw about his fæces and daub his hands and bedclothes with them.”

In what has been said above concerning the angular gyrus we have been alluding to a part of the cortex lying outside the occipital region (a purely artificial division), and yet functionally the two regions are evidently in the closest relation with one another and with the visual sense. Ferrier speaks, therefore, of an ‘occipito-angular region’ of the cortex.

Again, it should be noted that, what has been said above in regard to both the præ-frontal and the occipital cortical regions, is really applicable to the whole thickness of the brain in these situations, that is, to the præ-frontal lobes and the occipital lobes respectively.

3. Signs and Symptoms of Lesions in the Parieto-Temporal Region. This region includes the supramarginal lobule and angular gyrus, the convolutions of the temporo-sphenoidal lobe on its external and internal aspect, viz., the superior, middle, and inferior temporo-sphenoidal convolutions, the occipito-temporal convolutions (lingual lobule and fusiform lobule), the uncinate gyrus, and hippocampus major or cornu Ammonis.

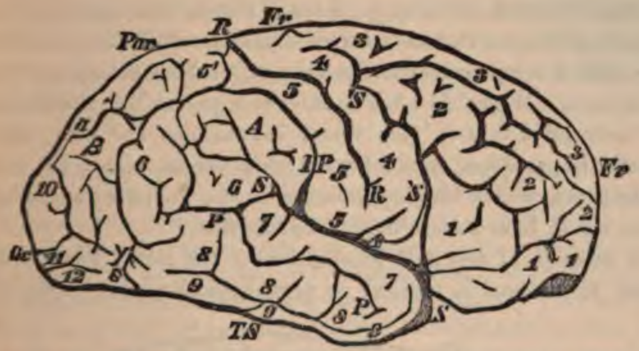

FIG. 34. PROFILE VIEW OF THE RIGHT CEREBRAL HEMISPHERE SHOWING ITS CONVOLUTIONS AND FISSURES [after Turner].

Fr Fr, Frontal lobe. Par, Parietal lobe.
Oc, Occipital lobe. TS, Temporo-sphenoidal lobe.
RR, Fissure of Rolando. SS, Sylvian fissure ; S'S' ascending limb of.
IP, Intra-parietal fissure ; PP, Parallel fissure.
1, 2, 3, Three tiers of frontal convolutions.
4,4 Ascending frontal, and 5,5, ascending parietal convolutions. 5', Outer part of postero-parietal lobule.
A, Supra-marginal lobule. 6,6, Angular convolution.
7,7, Upper, 8,8, middle, and 9,9, lower temporo-sphenoidal convolutions.
10, 11, 12, Superior, middle, and inferior occipital convolutions.
α, β, γ, δ, First, second, third, and fourth annectent convolutions.

This is commonly spoken of as the sensory region of the cerebral cortex, in opposition to the Rolandic or so-called motor region. What was said in the last section, however, will have sufficed to show that the occipital cortex has also sensory functions, at least so far as vision is concerned ; whilst, what has been said elsewhere (p. 113) leaves the question open to grave doubt whether the Rolandic area itself does not subserve sensory functions of the kinæsthetic order.

The writer was one of the first to call attention to the probable existence in the cortex of the cerebral hemispheres of what are now termed sensory or perceptive centres. Writing in Jan., 1869 ('Journal of Mental Science'), he said :—"though there may be much and compound overlapping of areas, and though the area pertaining to the impressions of any particular sense in the cerebral hemispheres may be a very extended one . . . still it may well be that certain portions of the surface of the cerebral hemispheres might correspond more especially to the maximum amount of nerve cells and fibres pertaining to some one or other of the special senses. . . . Just as certain of the senses contribute in a preponderating degree towards the building up of our mental impressions and their corresponding volitional results (*e.g.*, those of Sight, Hearing, and Touch), so we may imagine that these sense organs would be connected internally with a comparatively wide area of cortical substance in each hemisphere. It would be fair to infer as a probability, therefore, that the 'perceptive centres' for visual impressions, and also those for acoustic impressions, would have a wide-spread seat in the cerebral hemispheres, whilst those pertaining to the gustatory and olfactory senses would have a more limited distribution."

No mention of any such localisations in the cortex was, up to this period, to be found in medical or physiological works; although, as the writer then first attempted to show, such notions were capable of throwing much light upon cerebral physiology and upon certain defects of Speech resulting from disease of the brain ('Brit. and For. Med. Chir. Rev.,' Jan. and April, 1869). The writer's views in this direction were shortly afterwards endorsed and extended by Dr. Broadbent ('Med. Chir. Trans.,' 1872, p. 180).

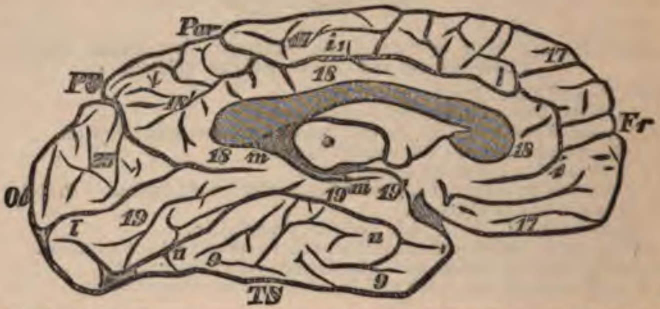

FIG. 35. INNER FACE AND TENTORIAL SURFACE OF THE LEFT CEREBRAL HEMISPHERE [after Turner].

Fr, Frontal lobe. Par, Parietal lobe. Oc, Occiptal lobe. TS, Temporo-sphenoidal lobe.
PO, Internal parieto-occipital fissure.
iii, Calloso-marginal, and l, calcarine fissure.
mm, Dentate fissure. nn, Collateral fissure.
17, 17, 17, Marginal convolution. 18, 18, Convolution of Corpus callosum. 18′, Quadrilateral lobule.
19, 19, uncinate convolution, of which 19′ is the crotchet, or recurved part.
25, Cuneus or occipital lobule.
9,9, Inner face of inferior temporo-sphenoidal convolution.

Ferrier, some years later, sought, by experimental evidence, not only to prove the existence but also to define the actual location of such sensory or perceptive centres ('Phil. Trans.,' 1875, p. 445) in the cerebral cortex, and he has undoubtedly thrown some most important light upon this very difficult subject. Whilst such a statement is only just, it must not be considered that I am prepared to accept all his results as final, or indeed to believe that some of the sensory centres are anything like so topographically limited as he represents them to be.

In his work 'The Brain as an Organ of Mind' (1880, pp. 522 and 530) the writer has referred to another possible interpretation of Ferrier's results. "These experiments of Ferrier are supposed by him to support the notion that perceptive centres limited in area, and topographically distinct from one another, exist in the cortex of the cerebral hemispheres. His facts, however, do not necessarily carry with them any such interpretation. They are quite capable of being explained in accordance with what we hold to be the more probable theory, viz., that such perceptive centres or mechanisms are diffuse in seat and interblended with others. This, indeed, has been pointed out by Prof. Croom Robertson, who says:—'so there is no intrinsic improbability—rather the reverse—in the view that impressions received by any organ of sense are all carried up first to a particular region of the cortical substance before they are brought into relation with other impressions and with motor impulses, or are otherwise elaborated in the brain. It may well be that there are special sensory organs in the brain-cortex, and that Dr. Ferrier has given the first rough indication of their locality.' Each set of sensory fibres may, in fact, direct themselves towards some particular part of the cerebral cortex, whence the fibres may diffuse themselves more or less widely. These 'first cortical stations,' or regions from which sensory fibres diffuse themselves in different directions, may have no real claim to be considered as 'centres,' and yet the same kind of results may follow from their destruction or stimulation as if they were real 'centres.' And, owing to the subsequent diffusion of the several kinds of fibres, other regions are not likely to be revealed by experimental investigation which would have any similar claims to be regarded as sensory centres."

What was said above has been in a measure justified by subsequent observations concerning the visual sense. Ferrier himself no longer limits its centre, as he did originally, to the angular gyrus; he now adds to it the cortex over the whole occipital region of the hemisphere. Perhaps, in the future, similar extensions may have to be made, enlarging the boundaries at present assigned by him in the cortex for the so-called centres for audition and touch.

As experimental investigations, made upon some of the lower animals by Ferrier, undoubtedly threw the first light upon the particular situations in the cortex the damage of which causes special impairment of this or that sense; and as, moreover, for various reasons, our knowledge derived from clinico-pathological data concerning these localisations is very defective, it will be best first of all to refer to these experimental results in regard to the localisation of the several so-called centres for touch, taste, smell, hearing, and vision.

The sense of Touch and common sensibility for all parts of one half of the body Ferrier believes to be localised in the hippocampal region of the opposite hemisphere of the brain—*i.e.*, in the hippocampus major and the overlying uncinate gyrus (Fig. 35).

The sense of Taste and the sense of Smell he localises in contiguous parts of the anterior extremity of the temporo-sphenoidal lobe, that is, in the so-called subicular region. Ferrier says in regard to this region :—"Destruction causes no motor paralysis, but is followed by loss of smell on the same side; and when the lesions invade not merely the subiculum but the neighbouring regions on one side, taste also is affected on the opposite side of the tongue. Bilateral lesions cause complete loss both of taste and smell."

The sense of Hearing is localised by Ferrier in the first or superior temporo-sphenoidal convolution. Referring to the effects of destructive lesions of one of these convolutions Ferrier says :—"though it is certain that hearing is at least impaired on the opposite side, the difficulty of ascertaining the condition of auditory perception in animals, when only one ear is affected, is such as to render it impossible to speak definitely as to the extent and duration of the affection of hearing; whereas when these centres are destroyed bilaterally, there seems to be a total loss of the sense of hearing; meaning by that auditory discrimination as contradistinguished from mere auditory reaction."

The sense of Sight is now localised by Ferrier in the occipito-angular region—that is, in the angular gyrus and occipital region combined.

The effects of stimulation of the angular gyrus on one side have already been referred to, in connection with the organic cause of lateral deviation of the eyes and head (p. 97).

In regard to the effects upon vision of destruction, partial or complete, of the occipito-angular regions Ferrier thus summarises his conclusions :—"Unilateral destruction of the cortex of the angular gyrus causes temporary abolition or impairment of vision in the opposite eye—not of a hemiopic character. . . . Destruction of the occipital lobe and angular gyrus on one side causes temporary amblyopia of the opposite eye and homonymous hemianopia of both eyes, towards the side opposite the lesion."

"Complete extirpation of both angular gyri causes for a time total blindness, succeeded by lasting visual defect in both eyes."

"The only lesion which causes complete and permanent loss of vision in both eyes is total destruction of the occipital lobes and of the angular gyri on both sides."

"As in none of the cases recorded either of partial unilateral or bilateral destruction of the occipito-angular region, were the amblyopic or hemianopic symptoms permanent, it is concluded that vision is possible with both eyes if only portions of the visual centres remain intact on both sides."

Even if we are to suppose, as seems probable, that something like the same set of facts concerning impairment of the different modes of sensibility would, under parallel conditions hold good for man, it must at the same time be confessed that little has been done as yet by clinico-pathological evidence to establish these localisations.

This is due to several circumstances, the nature of which varies somewhat for the different modes of sensibility.

In the first place, with the exception of touch and common sensibility, the various sensory endowments are not likely to be seriously interfered with by unilateral cortical lesions, after the first two or three days from the onset of a head affection, unless we have to do with bilateral, more or less symmetrical cortical lesions—which are, of course, met with only with extreme rarity.

Then, again, any sensory defect which may chance to be present during the first two or three days in the case of a unilateral cortical lesion is extremely apt to remain undetected, even if it be not actually unrecognizable. The general state of the patient at this stage of his illness (seeing that there may be some stupor, delirium, or, dulness of apprehension) is only too often such as to make examinations for the testing of sensibility either undesirable or little likely to be rewarded by any reliable results.

These are the two main general causes that tend to hamper the progress of our knowledge in this direction, that is, by clinico-pathological observations.

In regard to the reason why unilateral lesions of certain regions may produce profound and lasting defects in touch and common sensibility, and no profound or lasting results when occurring in regions associated with the functional activity of the more specialised modes of sensibility, I have elsewhere said ('Brain as an Organ of Mind, p. 489):—

"It is only in the sphere of the three higher senses, however, that a blending of the subjective accompaniments of impressions from the two sides of the body occurs so as to produce single Perceptions. An object which is smelt is perceived as one; a body which is seen is recognized as single; and, similarly, a sound, though stimulating both auditory organs, is heard as one sound. And, although we can localise gustatory impressions to one or other side of the mouth, when our attention is directed to the subject, we are not accustomed to do so, and there would be little use in making such discriminations. The case is altogether different, however, in regard to the sense of touch or common sensibility. By means of Smell, Sight and Hearing we are brought into relation with distant phenomena, but in the exercise of Taste or Touch there is actual contact with different portions of the extended surface of our bodies, and, therefore, in the latter case more especially, there ought to be, as there is, a thoroughly independent power of appreciating the impressions impinging upon each side of the body, and, indeed, of pretty accurately localising them."

Ferrier ('Localisation of Cerebral Disease,' p. 140) also makes some remarks bearing upon this point. He says:—"The facts of cerebral disease in general, and of cerebral hemianæsthesia in particular, would seem to show that in respect of tactile sensation there is less bilateral representation in each hemisphere than as regards the other forms of sensibility. For in cerebral hemianæsthesia, tactile sensation is always most deeply affected, and may still remain greatly impaired, after all the other forms of sensory impairment have disappeared. Hence, in the slighter forms of affection of the posterior third of the internal capsule, tactile sensation only may be impaired."

There is good reason, indeed, for believing that one cerebral hemisphere may take cognizance of the visual impressions made upon both eyes, as well as of the auditory impressions made upon both ears, since there are several cases upon record in which, although one hemisphere has been nearly totally destroyed, sight and hearing has remained almost unaffected on both sides. This has occurred in remarkable cases recorded by Andral, Abercrombie, Cruveilhier and others.

There seems to be only two possible modes of accounting for such facts.

We must either (a) suppose that each eye or each ear is (as the phrase goes) represented in each hemisphere—meaning by that, apparently, that from the nucleus or lower sensory centre in connection with each optic nerve, as with each auditory nerve, two sets of fibres proceed upwards to the cortex, the one set going to the hemisphere on the opposite side and the other to the hemisphere on the same side. This we may term the hypothesis of double structural representation.

Or (b) we must adopt some such explanation as I ventured to adduce in 1875 ('Paralysis from Brain Disease,' p. 107), when the following paragraph was written:—"We are compelled to fall back, as it appears to me, upon the same kind of reasoning as that employed by Dr. Broadbent to account for the exemptions from paralysis of the neck and trunk muscles on one side in cases of hemiplegia. Thus, in the case of motor phenomena, we find a unilateral outgoing stimulus impinging upon one side of double and united ganglia in the medulla or cord, and thence spreading to related muscles on both sides of the body. Conversely, in the case of sensory phenomena, we seem to have double ingoing impressions impinging upon sense organs on each side of the body, converging to double and united sensory ganglia, and these being taken cognizance of by a single cerebral hemisphere, when it happens that the other is seriously damaged . . . . . the perceptive action of the one hemisphere for sensorial impressions from the two sides of the body, would be likely to take place perfectly or imperfectly according to the frequency with which the two halves of the sense centres at the base of the brain are habitually stimulated in concert: because upon the frequency of this synchronism in action would depend the completeness of the commissural union existing between them. . . . . . . Physiologically however the case is quite different with regard to the sense of touch or common sensibility. In this mode of sentiency impressions derived from one side of the body engage our attention more frequently than simultaneous impressions on the two sides of the body; and even where the latter event occurs, the impressions on the two sides are discriminated from one another. In its ordinary activity, therefore, this is a mode of sentiency differing altogether from the others."

". . . what I have already pointed out is, that injury of the perceptive centres of one side, or of the fibres connecting them with the lower or basal ganglia, do not produce any hitherto appreciated effect on the senses of hearing, sight, smell, or taste, though it does produce some effects upon common or tactile sensibility. The result is, however, in each instance altogether different if we have to do with damage to the basal nuclei ('sense centres'), or to the nerve trunks connecting them with the peripheral parts or sense organs proper."

This we may term, for the sake of distinction, the hypothesis of double functional representation.

It remains now to say a few words concerning damages occasioned by disease or injury, as occurring in the human subject, to the different cortical regions of the temporo-sphenoidal lobes which have been above referred to in connection with the several sensory endowments, and to the conditions under which they are severally most prone to occur.

Hippocampal region.—As yet no cases of gross lesion, such as softening or hæmorrhage, limited to the hippocampal region on one side, have been carefully observed and placed on record. This is one of the gaps that will have to be filled up in the future. Thus, although experiments upon animals seem to show, as above indicated, that unilateral lesions of this region of the cortex may produce lasting loss or impairment of sensibility on the opposite side of the body, we know nothing of any such result from clinico-pathological observations upon man. In any future observations that may be made, it must be well borne in mind that the posterior third of the hinder segment of the internal capsule (that is, the portion in which all the sensory fibres are massed together) lies in close proximity with the hippocampal region. Its integrity, as well as its freedom from pressure, must therefore be established before in any case an anæsthesia of the opposite side of the body can be safely referred to a lesion in the hippocampal region (see Fig. 220 Ross).

> Ferrier ('Localisation of Cerebral Disease,' p. 126) says:—"Of lesions specially confined to the hippocampus, I have not been able to find any on record, except those in respect to degeneration or sclerosis of the hippocampus in chronic epileptics. Bouchut, in twelve out of forty-three cases which he examined, noted the existence of sclerosis in one or both hippocampi, but he did not attach any special importance to this; as induration of the brain in chronic epilepsy he looked upon as a general affection of which this was only a local manifestation. This condition of the hippocampus has been observed also by Casuvielh, Foville, Lélut, Delasiauve, and Bourneville, in epileptics. In 1868 Meynert called special attention to this degeneration of the hippocampus in epileptics, giving nineteen cases in which one or other hippocampus was indurated or atrophied. Meynert without looking upon this as *the* cause of epilepsy, thought that there was some special relation between this degeneration and the lesion on which epilepsy depended. In a recent paper Hemkes states that he has seen atrophy of the hippocampus in only six out of thirty-four cases of chronic epilepsy. Beyond the fact of the existence of such degeneration in epileptics, we have no record of the exact symptoms in the cases in which it was found."

Subicular region.—These regions of the hemispheres are occasionally damaged in cases in which a person falls from a height upon his head, and yet does not receive injuries sufficient to prove fatal. The tips of both temporal lobes may, in such a case, be bruised and lacerated simultaneously, with the effect, it is presumed, of more or less completely destroying the sense of smell and hearing on both sides. Ferrier refers to a case that came under his own observation in which this seemed to be the most probable interpretation. The essential peculiarity which would point to the probability of such an occurrence is the simultaneous loss of taste as well as smell, dating from a traumatism of the kind above referred to—the loss of taste being real and not a mere diminution in the ability to appreciate flavours such as may result from loss of smell alone (Part III, A). This double loss could otherwise only be explained by the supposition that widely separated nerves (having, on account of their respective situations, no special tendency to be affected together) had nevertheless been simultaneously involved—which would, of course, be rather improbable.

To warrant the diagnosis of lesion in, or damage to, the subicular regions, it is needful that we should meet with the double simultaneous loss of smell and taste, since, as pointed out by W. Ogle, many cases are now on record of loss of smell with merely slight impairment of taste, as a result of blows on the head, especially on the vertex or occiput. As he pointed out, this may be due to damage by counter-stroke to the olfactory nerves, bulbs, or tracts. In some of these cases there may be a loss of smell only, whilst in others there may, in addition, be diminutions to various degrees (not loss) of the sense of taste, due merely to the loss of the sense of smell as above indicated. (See Part III, A).

Concerning the effects of unilateral lesions of the subicular region, owing to disease (softening, abscess, tumour) or to traumatisms, we as yet know nothing definite. This is one of the points that requires to be solved by future observation—but it is just one of those points in which we may have to wait long before any settlement is arrived at. We can only say that, in accordance with the experimental indications afforded by Ferrier, there should be loss of smell on the side of lesion and loss of taste on the opposite side.

From what is said below, however, it will be seen that Ferrier indicates the possibility of another result, viz., mere diminution of the sense of smell on the two sides, with loss of taste on the side opposite to the lesion. But a mere diminution of the sense of smell on the two sides would be, practically, a defect incapable of detection in nine patients out of ten, the degree of whose previous endowments in this respect would, in all probability, have been unknown.

"As regards smell," Ferrier says, "there seems to be some discrepancy between my localisation of the olfactory centre and the facts of cerebral hemianæsthesia. I find that destruction of the subicular region causes loss of smell on the *same side*; while in hemianæsthesia the impairment of smell is on the side *opposite* the cerebral lesion. I have endeavoured to account for this by the fact discovered by Magendie, that abolition of the *common* sensibility of the nostril by section of the sensory branches of the fifth nerve causes loss of smell; and as in hemianæsthesia the sensibility of the mucous membrane of the nostril is lost, so we may consider this to be a sufficient cause of the unilateral anosmia. I see no reason to doubt the validity of this explanation but I would supplement it by another consideration. Though he outer root of the olfactory tract can be directly traced to the subiculum of the same side, it is not unlikely that the inner root passes on to the opposite hemisphere with the other sensory tracts; and hence each hemisphere may maintain a bilateral relation with the organ of smell. If this were so, then the partial impairment of smell, which would result from lesion of the special sensory paths of the opposite hemisphere would be rendered more complete by the simultaneous abolition of common sensation in the nostril." Anatomical evidence of this distribution of the inner root of the olfactory nerve is not yet forthcoming.

Region of the Superior Temporo-Sphenoidal Convolution.—Unilateral lesions in this situation have never, as yet, been known to give rise to deafness or any noticeable diminution of the power of hearing. It seems possible that an acute lesion occurring in this situation might produce some deafness on the opposite side for a few days from the onset of the lesion. The patient might, however, within such a period, not be sufficiently conscious, or sufficiently quick of apprehension, to make an examination as to possible diminutions in degree of sensibility, of this or that kind, yield any very satisfactory results.

Bilateral lesions in the first temporo-sphenoidal convolution can be expected to occur only with extreme rarity. When they do present themselves there ought, in accordance with Ferrier's results, to be the production of more or less complete deafness. I am not aware that any such cases are as yet on record.

Subjective noises (such as may occur in the initial stages of epileptic attacks), as well as hallucinations of hearing, should probably be regarded as results of abnormal nerve actions taking place in this or in some other cortical station connected with the sense of hearing.

Lesions principally occupying this region of the upper temporal convolution on the left side were found by Wernicke to be associated

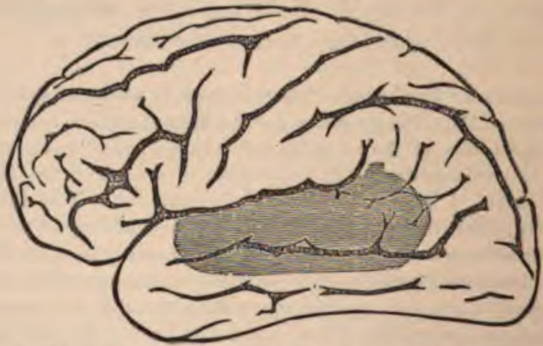

FIG. 36. DIAGRAM SHOWING THE SEAT OF LESION IN WERNICKE'S CASE OF WORD-DEAFNESS

during life with an amnesic defect known as 'word deafness.' And this, so far as it goes, gives support to Ferrier's view that the upper temporal convolution is at least intimately concerned with the sense of hearing. In this state the individual is able to understand printed or written words which he sees, and he is not deaf in the opposite ear—it is rather that he does not understand or apprehend the meaning of spoken words; he hears them as sounds, but they are to him sounds which convey no meaning. It may be supposed, therefore, that a special part of the cortex in which words are accustomed to be revived in memory (as sounds), and which, for the sake of convenience, may be spoken of as the auditory word-centre, has been damaged; or else we must suppose that a lesion invading the cortical auditory centre on the left side simply renders the performance of one of the most specialised functions of this auditory centre impossible. In regard to lower, or less specialised, uses of the auditory centre, there may be no abolition of auditory perception and discrimination.

It should be borne in mind that even total deafness in the right ear (the nerve fibres from which are in relation with the auditory cortical centres of the left hemisphere) does not carry with it any approach to word-deafness as above described. We must suppose, therefore, in such a case that the intact cortical auditory centre of the left side is called into activity through the corpus callosum by stimuli from the right cortical centre (which is in connection with the healthy ear). This seems the only way of accounting for the exemption from 'word-deafness' of patients who are the subjects of right-sided deafness.

From what has been said above, it will follow that if a person who is deaf on the right side, becomes the subject of a cerebral lesion that involves (a) the right superior temporal convolution, or (b) the portion of the corpus callosum which serves to connect it with its fellow convolution in the opposite hemisphere, he would thereby be reduced to a state of word-deafness.*

The presumption at present is, that a person who is not deaf on either side would not be reduced to a condition of word-deafness by a lesion in the right superior temporal convolution—though possible exceptions to this might be met with in the case of patients who are markedly left-handed.

Occipito-angular Region.—The effects upon vision of lesions in this region of the cortex have already been fully discussed in the section on Amblyopia and Hemiopia (pp. 143-146), and have been again referred to when speaking of the Occipital Region of the cortex. Nothing further need be said upon the subject here, except in relation to the production of a peculiar amnesic state to which the term 'word-blindness' has been applied. The state itself is comparable with an analogous defect in the sphere of another sense, viz., 'word-deafness.' When 'word-blindness' exists the individual is unable to comprehend the meaning of written and printed words (though he may be able to recognize and distinguish common objects perfectly), while he is at no loss to understand oral speech—that is, the speech which he has to appreciate in the main through his auditory centres. It is at present supposed, upon the basis of a case carefully observed during life and examined after death by Broadbent, that the condition may be produced by a lesion occupying the site of the angular and supra-marginal gyri of the left side. This was the particular region damaged in Broadbent's case (Fig. 37) in which there was not only evidence of the existence of 'word-blindness,' but also of some defect in the commissural fibres uniting the visual with the auditory word-centres.

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* For reference to a possible case of this kind see the writer's work, 'The Brain as an Organ of Mind,' p. 632, where there are some observations concerning the interpretation of a remarkable case recorded by Dr. Banks.

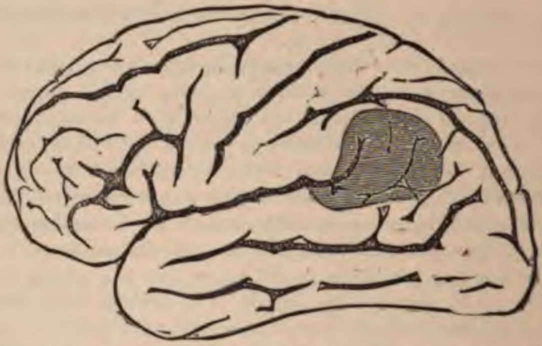

FIG. 37. DIAGRAM SHOWING THE SEAT OF LESION IN BROADBENT'S CASE OF WORD-BLINDNESS.

It should be borne in mind that even total blindness in the right eye (the nerve fibres from which are principally in relation with the visual cortical centres of the left hemisphere) does not carry with it any approach to word-blindness as above described. We must suppose, therefore, in such a case, that the intact cortical centre of the left side is called into activity by the right cortical centre (through the intervention of the corpus callosum) which is in connection with the healthy eye. This would seem to be the only way of accounting for the exemption from word-blindness of patients who are merely the subjects of right-sided blindness, on the supposition that an uncomplicated cross relation exists between the eyes and the cerebral hemispheres. The doubt existing in regard to this point has, however, been already fully referred to (p. 136).

From what has been said above, it will follow that if a person who is blind on the right side becomes the subject of a cerebral lesion which involves (a) the right angular and supra-marginal convolution, or (b) the portion of the corpus callosum which serves to connect these with their fellow convolutions in the opposite hemisphere, he would probably be reduced thereby to a state of 'word-blindness.'

The presumption at present, however, is that a person who is not blind on either side, would not be reduced to a condition of word-blindness by a lesion in the right angular and supra-marginal gyri—though possible exceptions to this might be met with in the case of patients who are markedly left-handed.

From what has been said it seems clear that lesions in the Parieto-Temporal Region of the cortex give rise to but few symptoms which have any localising value. Lesions confined to this region of the cortex cause neither paralysis nor convulsions; and they do not, as a rule, when unilateral, give rise to any recognizable impairments of sensibility. When occurring in the left hemisphere, however, a lesion involving the upper temporal convolution will, in all probability, cause word-deafness; whilst a lesion occupying the angular and supra-marginal gyri is just as likely to cause word-blindness.

Lesions on the confines of the two regions above specified will, also, very probably cause cases of Amnesia like those which I have described as being due to lesions of the '*Commissures between the Auditory and the Visual Centres*' ('Brain as an Organ of Mind,' p. 640).

Lesions in the angular gyrus have also sometimes been associated with lateral deviation of the head and eyes (see p. 97).

Symmetrical bilateral lesions of different parts of the cortex of this parieto-temporal region, rare as they are, will, when they occur, cause, according to their precise locality and extent, various sensory paralyses, and in some cases impairments of intellectual expression.

Even in the case of the many unilateral lesions in this parieto-temporal region (especially abscess and tumour), in which symptoms having a localising value are absent, there are, nevertheless, as a consequence of the lesion, the general symptoms indicative of an intra-cranial growth or swelling.

4. Signs and Symptoms of Lesions in the Rolandic Region.—This is what is commonly spoken of, after Ferrier, as the 'motor area' of the cortex. It is however much better, in our opinion, to speak of it either as the 'Rolandic region,' or more simply still as the 'excitable area' of the cortex, since these terms carry with them no theory as to the explanation of the effects of disease or experiment upon this part of the cortex—this being a matter still open to considerable doubt (p. 113).

The region in question includes the bases of the three frontal convolutions, the convolutions bounding the fissure of Rolando (ascending frontal and ascending parietal), the parietal lobule, and a part of the marginal convolution on the inner surface of the hemisphere (p. 165). This region corresponds very closely with the shaded areas in Figs. 38 and 39.

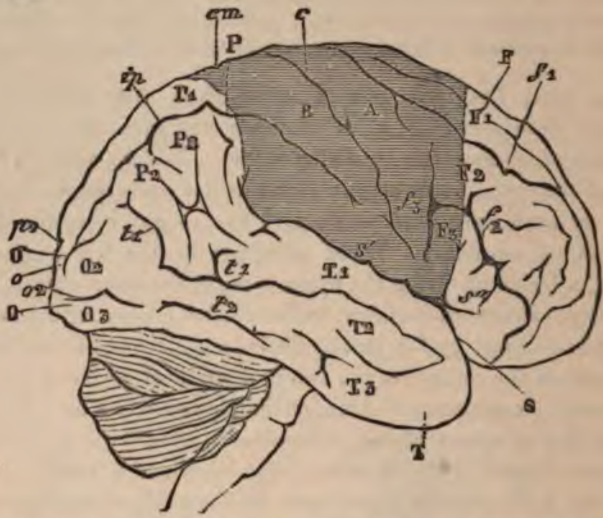

FIG. 38. DIAGRAM OF THE OUTER ASPECT OF THE RIGHT HEMISPHERE, ROUGHLY SHOWING THE BOUNDARIES OF THE ROLANDIC OR EXCITABLE AREA (THE SHADED REGION). The shaded region in this figure extends posteriorly a little beyond the Rolandic area below, and a little short of its confines above.

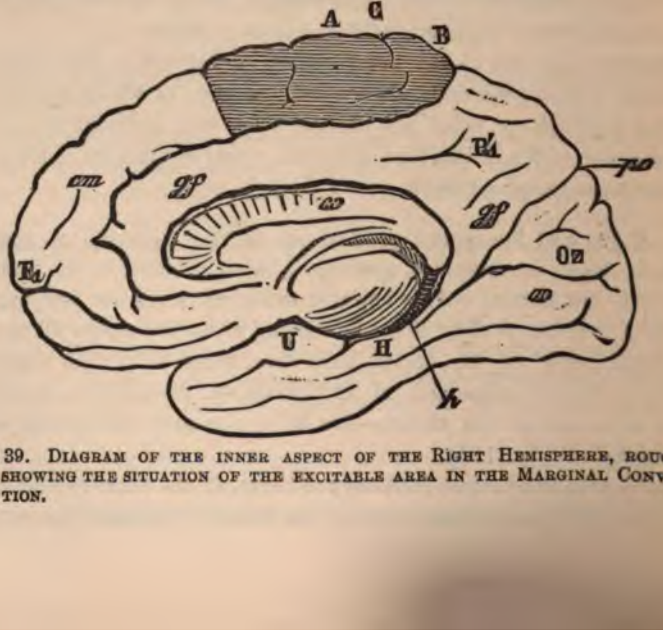

FIG. 39. DIAGRAM OF THE INNER ASPECT OF THE RIGHT HEMISPHERE, ROUGHLY SHOWING THE SITUATION OF THE EXCITABLE AREA IN THE MARGINAL CONVOLUTION.

Facts of importance concerning the Blood-supply of the Excitable Area of the Cortex.—The part of the excitable area belonging to the inner aspect of the hemisphere (posterior half of marginal convolution) is supplied by the middle and internal branch of the frontal artery, whilst the part on the outer aspect of the hemisphere is supplied by branches of the Sylvian or middle cerebral artery.

This separateness of the vascular supply of these two regions is worthy of remark, especially in conjunction with the fact that softenings are much more common in the territory of the middle cerebral than they are in that of the anterior cerebral. Seeing that one of the convolutions with which we are now concerned, viz., the marginal convolution, is supplied by a branch of the anterior cerebral, and that this is principally in relation with the trunk muscles of the opposite side of the body, the exemption of these muscles from paralysis in cases in which occlusion of the middle cerebral artery causes softening involving more or less of the excitable area on the outer surface of the hemisphere supplied by the Sylvian artery, is only what might now be expected. Accordingly, in regard to hemiplegia produced by such causes, it would be unnecessary to have recourse to Broadbent's hypothesis to account for the exemption from paralysis of the trunk muscles.

The main portion of the Rolandic region is supplied by three branches of the Sylvian artery each of which nourishes a special area (Fig. 28). The artery may be occluded (by embolism or thrombosis) in its main trunk or in one or more of its separate offsets. In the latter case we may have monoplegias of different kinds according to the particular branches affected; whilst, where the whole of the cortical branches, or the main trunk, is involved we may have a complete hemiplegia of the opposite side (the trunk muscles and other bilaterally acting muscles only being exempt), with or without aphasia, according as the left or the right hemisphere is affected in right-handed individuals (or *vice versâ* for left-handed persons). All this may occur from an occlusion of the middle cerebral beyond the first part of its course ($\frac{2}{3}$ of an inch), that is, beyond the part from which the twigs of the basal system are given off to the internal capsule and to the corpus striatum (and, consequently, in the absence of any subsequent softening of these parts). The clinical phenomena are not, however, appreciably different when this first part of the Sylvian artery becomes occluded—even though this entails softening of the motor segment of the internal capsule and of the greater portion of the striate body, as well as of the cortex in the Rolandic area.

The first of the cortical branches of the Sylvian artery is supplied to the posterior part of the third frontal convolution, and if this vessel only becomes occluded or becomes the seat of an enduring functional spasm, on the left side, we may have as a result an aphasic condition without other paralytic complication. Similarly, the separate implication of other branches of the Sylvian artery will lead to other forms of monoplegia, to which reference will presently be made.

In reference to lesions of this region of the cortex in particular it is especially needful to bear in mind that they may be of two kinds—

either destructive or irritative. And yet, as Ferrier says, "a complete practical separation is not always possible, inasmuch as lesions which result ultimately in total disintegration and paralysis, are not unfrequently associated at times with irritative or convulsive phenomena limited or more or less generalised."

This distinction, supported as it is by clinical observation, derives its main support from the results of experiments upon animals. Speaking of the Rolandic area in the monkey, Ferrier says:—"In this region are situated certain definable areas, stimulation of which by the electric current gives rise to certain definite movements on the opposite side—viz., of the leg and hand, facial, oral and lingual muscles; and destruction of which causes paralysis of all these movements if the entire region be destroyed; limited or dissociated paralysis, if individual areas only be destroyed—the paralysis in this case being confined to those movements which are excited by irritation of the same."

The fact that irritation of a given portion of the Rolandic area produces contraction of a certain set of muscles, and that a destructive lesion of the same portion of the Rolandic area gives rise to a paralysis of this set of muscles, makes it quite unnecessary for us to go over the diagnostic indications from the double point of view of irritative and destructive lesions. We shall, therefore, after referring to injury of the area as a whole, confine ourselves to the specification of the different kinds of monoplegiæ which may result from destructive lesions of the different portions of the Rolandic area, with the understanding that corresponding monospasms, may be induced by slighter disease in the same regions, and that, as a matter of fact, such monospasms do often precede the establishment of a monoplegia. At other times we have a kind of coincidence of the two functional states—that is to say, the monoplegia may be partial, and at intervals there may be attacks of monospasm, invading the same muscles, in the first instance at least, even though the spasm may spread so as to invade other muscles in later stages of the same fit. Again, we may have a monoplegia of one kind complicated with a monospasm of a different order, in cases where destructive and irritative lesions coexist in different portions of the Rolandic area.

It is important to bear in mind, moreover, that typical monospasms (monospasms, that is, which always begin in the same way) possess a localising value, similar in kind if not in degree, to that which may be derived from corresponding monoplegiæ due to destructive lesions.

Destructive lesions of the Rolandic Area may be either (1) general, or (2) partial.

1. **General lesions.**—These, as already indicated when speaking of the vascular supply of the Rolandic area, are only with extreme rarity likely to be absolutely complete. By far the most frequently, the general lesion is limited to the territory of the Sylvian artery, and thus does not involve the part of the marginal convolution included within the excitable area, which, as above indicated, is supplied by a branch of the anterior cerebral.

There is thus produced a hemiplegia of the ordinary type in which, without affection of sensibility to any very appreciable extent, there is paralysis of voluntary motion on the opposite side of the body, with the exception (p. 172) that "associated, alternating, or bilateral movements are more or less spared." This paralysis is frequently associated with rigidity or convulsive spasms in the paralysed parts, particularly in the early stage; and, if destruction of the cortical substance be complete, the paralysis is permanent, and is, after a time, followed by late rigidity and secondary sclerosis of the lower motor tracts in the brain and spinal cord.

Where the lesion involves the left Rolandic area, in an ordinary right-handed individual, the hemiplegia is associated with Aphasia.

It will be well to cite two or three instances of general lesions in the Rolandic area of the cortex, giving rise to a permanent hemiplegic condition. We select, therefore, three of the most typical of those referred to by Ferrier.

A case is reported by Gliky of unilateral convulsions of the left side followed by complete left hemiplegia without loss of sensation, in which, after death, a caseous degeneration was found involving the following parts in the opposite cerebral hemisphere, viz., the ascending frontal and bases of the three frontal convolutions, the ascending parietal and postero-

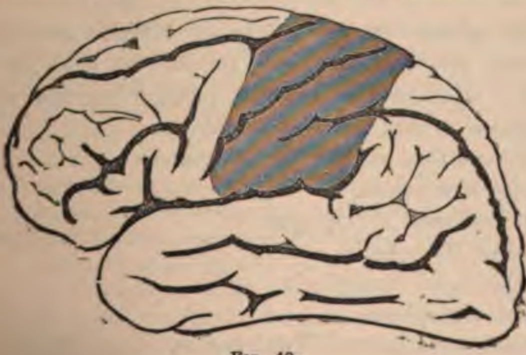

FIG. 40.

parietal lobule, together with the corresponding internal aspect of this region, or paracentral lobule (Figs. 38 and 39).

Lepine has recorded a case of right hemiplegia, without affection of sensation, of six years' duration, caused by yellow softening of the ascending parietal convolution in its whole extent, with partial affection of the ascending frontal, posterior digitations of the island of Reil, and anterior part of the superior and inferior parietal lobule of the left hemisphere (Fig. 40). The ganglia were intact. Secondary degeneration was traced in the left side of the pons Varolii and left pyramid.

A case is recorded by Charcot and Pitres of right hemiplegia with aphasia, of one year's duration, and accompanied by late rigidity of both limbs, more particularly of the arm. A patch of yellow softening was found involving the whole of the ascending frontal and base of the third frontal convolution, the whole of the ascending parietal, together with the inferior parietal lobule and two posterior digitations of the island of Reil in the left hemisphere (Fig. 41). The ganglia were normal. Secondary degeneration was evident in the crus, pons, and pyramid on the same side.

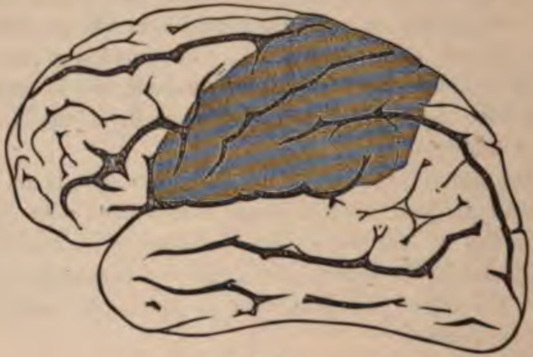

FIG. 41.

2. Partial lesions.—Partial lesions in different portions of the Rolandic area, give rise, as a rule, to different local paralyses or monoplegiæ. Of these the following varieties together with their regional indications must now be referred to:—

(a). Unilateral Oculo-Motor Monoplegia.
(b). Crural Monoplegia.
(c). Brachio-Crural Monoplegia.
(d). Brachial Monoplegia.
(e). Brachio-facial Monoplegia.
(f). Facial Monoplegia.
(g). Aphasia (Oro-Lingual Hemiparesis).

Still it happens occasionally, that a lesion involving only a part of the Rolandic area, is nevertheless associated with a complete hemiplegia. In such a case we must assume the existence of a functional disturbance over an area wider than that of the lesion. This is, of course, more especially apt to occur in the early stages of cases in which the onset of the lesion is sudden. Still it is not always so. Cases are on record in which with a limited chronic lesion in the Rolandic area, there has, nevertheless, been established a pretty complete hemiplegia of the ordinary type—that is, involving the limbs and face, but without sensory impairment. It is possible that other small undetected lesions may have existed in these cases.

(a). *Unilateral Oculo-Motor Monoplegia.*—This subject has already been considered in the section concerning Conjugate Deviation of the Eyes and Head, to which the reader is now referred (p. 95).

(b). *Crural Monoplegia.*—In considering the question of this and of other limited paralyses from the point of view of localisation it must be borne in mind that our knowledge on the subject is only now being established, and that the facts supplied by experiment are, for the present, rather more definite and precise than those which have been acquired by clinical medicine. So far as they go, however, the two sets of facts seem to be pretty closely in accordance with one another.

Still, as Ferrier himself points out, doubts and difficulties of various kinds beset us, in our endeavours to arrive at precise and reliable localisations. He says, in reference to this point ('Localisation,' p. 65):—"It is a question by no means easy to answer *à priori*, how far the movements of the human leg can be compared with those of the leg-arm and foot-hand of the monkey; or what is the representative in man of the centre for the tail which in the New-World monkeys plays the part of a hand. We must, therefore, be cautious in drawing conclusions as to the exact positions of the arm and leg centres in man from considerations merely of anatomical homology. And there is reason for exact and careful analysis of the movements which are affected, or more particularly affected, in any given case of crural monoplegia of cortical origin, for on this may depend the exact regional diagnosis. . . . . Clinical evidence in favour of a distinct centre or centres for the leg, clearly differentiated from those of the arm, is not as yet very extensive. We have many cases on record in which the leg and the arm have been paralysed together—brachio-crural monoplegia—an association easily accounted for by the close relation of the leg and arm centres to each other. Still there are some cases in which the leg *only* has been paralysed; and in others, in which leg and arm have been ultimately conjointly affected, the paralysis has shown itself *first* in the leg. This latter fact has an important bearing on the question of a distinct leg-centre, and its exact situation. A few cases are on record of paralysis occurring in one or both legs from injury to the vertex in the parietal region; but we cannot exercise too much caution in the inferences we draw as to the seat of lesion in such cases. The researches of Duret ('Traumatismes Cérébraux,' Thèse., 1878) have shown that local and general spinal paralyses may result from bulbar and spinal lesions owing to sudden displacement of the cerebro-spinal fluid in consequence of blows on the head."

Elsewhere Ferrier says:—"I think however that though the exact limits of the leg centres are not defined, the clinical evidence points to a position closely in harmony with that defined in the brain of the monkey."

The indications furnished by experiment lead us to believe that the excito-motor centre for the movements of the lower extremity are situated around the upper extremity of the fissure of Rolando, occupying the upper extremities of the ascending frontal and ascending parietal convolutions, as well as slightly forward into the superior frontal gyrus and slightly backwards into the parietal lobule. According to Horsley and Schäfer, also, it "dips largely over into the marginal convolution opposite the upper end of the fissure of Rolando."

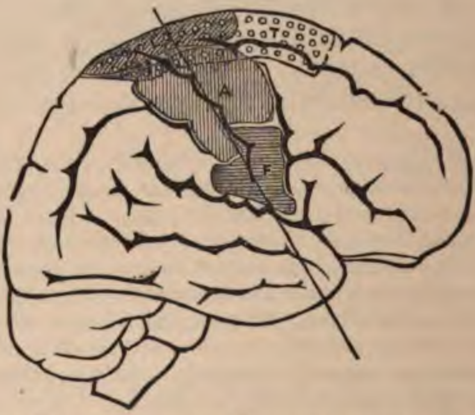

FIG. 42. RIGHT HEMISPHERE OF THE CEREBRUM SHOWING THE SITUATION OF DIFFERENT CENTRES IN THE EXCITO-MOTOR AREA OF THE CORTEX, ARRANGED ROUND THE FISSURE OF ROLANDO, AS INFERRED FROM EXPERIMENTS ON MONKEYS [after Horsley].

F, facial centre. A, centre for upper limb.
L, centre for lower limb. T, centre for trunk muscles.
(This latter centre being really on the mesial surface of the hemisphere, is represented at its border by dotted lines. The straight line represents almost the plane of section shown in Fig. 14).

The clinical cases (whether of monospasm or monoplegia) which can be brought forward in illustration of this localisation are undoubtedly very rare; it may be well, therefore, to cite some of those which have been adduced in support of it—taking them merely for what they are worth.

Hitzig quotes from Löffler the case of a Danish corporal who was struck by a shot at the superior and posterior extremity of the left parietal bone, close to the sagittal suture, which, by reference to Fig. 33, may be seen to overlie the postero-parietal lobule. The right leg was immediately paralysed. The right arm became affected on the seventh day. On trephining, recovery took place—the arm first and then the leg. Ferrier justly observes, "This may fairly be taken as a cortical lesion, as the subsequent affection of the arm is in accordance with extension of softening to neighbouring centres; a feature so characteristic of cortical lesions."

Rendu also records a case of paralysis of the right leg followed by paresis of the right arm, which gradually increased. In the parietal convolutions of the left hemisphere, close to the longitudinal fissure, was a zone of exudation and interstitial hæmorrhages, affecting both the cortex and the medullary substance to a considerable depth, but not extending to the basal ganglia. In the parietal convolution (exact position not stated) was found a caseous nodule of the size of a nut.

Gougenheim has recorded a case of a man aged 45 who was seized with paralysis of the left leg, without affection of sensation. A few days after, the paralysis increased, and the left arm became similarly affected. Coma came on and death. Diagnosis was made of a lesion of the upper extremity of the ascending frontal. The necropsy revealed a lesion, tuberculo-meningeal, of the ascending frontal, but especially involving the paracentral lobule (that is the portion of the marginal convolution opposite the ascending frontal and parietal convolutions).

One of the best cases has, however, been recorded by Ferrier himself ('Brain,' April, 1880). A man with some phthisical symptoms began to complain one day of paresis of the left leg; this increased, so that two days later the leg became completely paralysed. A few days after, he began to complain of weakness in the left arm, and within three days more this also had become completely paralysed. At this time both limbs were flaccid and powerless, so that there was complete loss of volitional power over them. There was no trace of facial paralysis, and the tongue was protruded straight. There was an increased knee-jerk in the left leg. Sensibility was unimpaired in the limbs or elsewhere. The temperature was ·8° F. higher in the axilla of the paralysed than in that of the non-paralysed side. About a week after the onset of the paralysis in the arm, the patient had a series of convulsive movements of the left leg and arm, commencing in the leg and lasting half an hour. He died in one month from the onset of the first signs of weakness in the leg. At the autopsy the following condition was found:—"The pia mater was everywhere normal and separated readily from the cortex except at one spot. This was situated at the upper margin and internal aspect of the right hemisphere on both sides of the fissure of Rolando, where the pia mater merged into a caseous adhesion not appreciably elevated above the rest of the cortex." Further careful examination showed the exact position and extent of the lesion to be as follows:—"The pia mater was adherent and studded with minute tubercular foci, of a yellowish colour, on the internal aspect of the hemisphere, at the point where the fissure of Rolando terminates. The adhesion was of a quadrilateral shape, measuring 1 inch antero-posteriorly and ¾ inch vertically. . . . Doubling over the margin of the hemisphere the adhesion separated the extreme upper lips of the fissure of Rolando from one another, and descended along the bottom of this fissure, coming to a point an inch below the margin of the hemisphere. The whole area and shape of the lesion may be likened to a triangle, the base of which would measure 1 inch, and the height 1¾ inch or 2 inches at the utmost, applied with its base at the internal termination of the fissure of Rolando, and doubled over the margin with its apex directed along the bottom of this sulcus." The lesion was also found to be strictly limited to the cortex, and to be "most advanced internally and at the extreme upper margin of the hemisphere." The rest of the brain was normal throughout. The lungs were studded with miliary tubercles.

In some other recorded cases bearing upon the production of a crural monoplegia, the **marginal convolution** has also been the seat of lesion, which, though in part containing centres in relation with leg movements, is also, according to Horsley and Schäfer, especially in relation with trunk muscles. In the three cases belonging to this category to which we are now about to refer, no mention is made of the existence of paralysis of the trunk muscles on the opposite side of the body. This is not surprising, seeing that these bilaterally acting muscles may, in all probability, in accordance with the hypothesis of Broadbent, be called into activity by the motor stimuli emanating from the sound hemisphere. Lesions of both marginal convolutions would therefore be necessary, in accordance with this view, in order to bring about paralysis of the trunk muscles (see p. 174)—a combination of the greatest rarity. Evidence concerning the implication of centres in relation with the trunk muscles in the marginal convolution should be sought, therefore, from unilateral lesions which are of an irritative rather than from those which are of a destructive type. Irritation of these centres in one marginal gyrus, ought, if Broadbent's view be correct, to produce bilateral convulsions of the trunk muscles. These are points to which the attention of future observers must be directed.

Ferrier refers to a case published by Haddon, which, though one of tumour, has considerable value in relation to the existence and mode of causation of crural monoplegia. "The patient began first to have tingling in the left leg, followed by paresis of this limb, gradually increasing, and continuing restricted to the left leg for five months. Then the left arm became weak. After occasional attacks of rigidity and twitching, the arm and leg became both completely paralysed. Shortly before death signs of weakness showed themselves also in the right leg. After death a tumour, three inches in diameter, was found growing from the dura mater, and pressing perpendicularly downwards on the region included in the upper extremity of the ascending frontal, ascending parietal, and postero-parietal convolutions (Figs. 43 and 44). The tumour had grown downwards as far as the

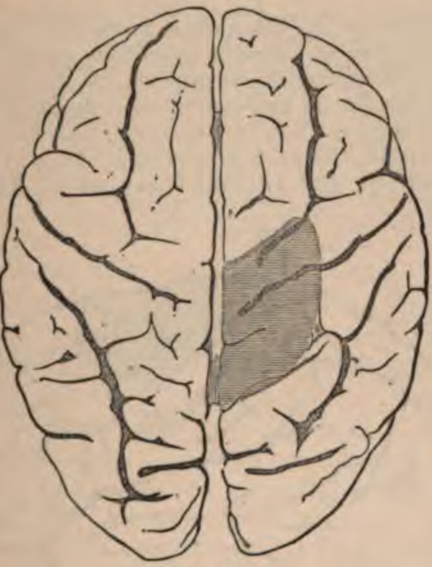

FIG. 43.

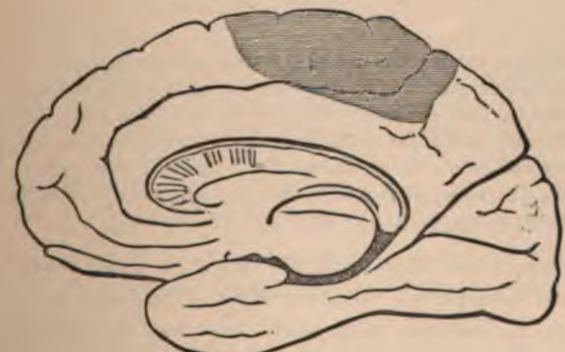

FIG. 44.

floor of the lateral ventricle, compressing the brain substance in its course. It also impinged on the other hemisphere. The point of origin of this tumour, and the mode of growth, warrant the conclusion that the paresis of the left leg, which was the first, and for a long time the only motor symptom, was due to the affection of the cortex in the region where experiments in the monkey would lead us to localise the leg centres."

Two cases have been reported by Bourneville, which also tend to throw light upon the problem as to the part of the cortex that contains the excito-motor centres for the leg, though they are far from being uncomplicated cases of crural monoplegia.

The first is that of a girl, aged 18, who had become hemiplegic on the left side at the age of four. She was subject to epileptiform attacks, beginning in the paralysed left leg. There was no facial paralysis. At the autopsy a patch of degeneration was found in the right hemisphere, occupying the upper half of the ascending frontal, the bases of the first and second frontal, the anterior part of the postero-parietal lobule and the whole of the internal aspect of these regions (Fig. 45).

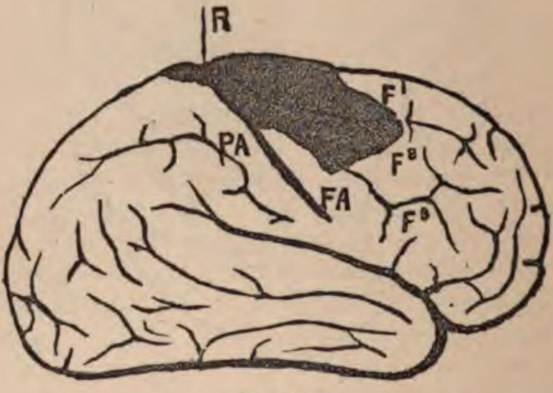

FIG. 45.

The second case was likewise one of infantile hemiplegia, followed by partial epileptiform convulsions. The spasms were limited to the right leg at first, but gradually invaded the right arm and the right side of the face. After death a lesion was found, closely according in site with the diagnosis which had been made during life, that is, occupying the upper extremity of the ascending frontal and parietal convolutions, and the internal surface corresponding, or the paracentral lobule.

To the cases above cited I will add two of crural monospasm, which have been placed upon record by Hughlings Jackson, and which are of considerable value in reference to the localisation now under discussion.

In the first case, fits began almost invariably in the right leg and were frequently limited to it. The leg began to become weak, and more so after each fit, the paresis deepening ultimately into a permanent paralysis. In the last stages, signs of a more general affection of the left hemisphere, including aphasia, manifested themselves. A tumour was found at the upper and posterior part of the left frontal lobe, about two inches in diameter, bounded posteriorly by the fissure of Rolando, and extending forwards into the posterior part of the first and second frontal convolutions.

In the second case, there were convulsions beginning in the left great toe, often confined exclusively to the leg, and followed ultimately by paresis of the left foot. This patient also had paralysis of the right third nerve. After death a syphilitic lesion was found at the upper part of the ascending parietal convolution, extending over part of the upper end of the ascending frontal and over adjacent parts of the parietal lobule of the right hemisphere. On the right third nerve a tumour of the size of a pea was found. The lesion here almost exactly corresponded in site with that assigned by Ferrier to the leg centres.

(c). *Brachio-Crural Monoplegia.*—The immediate contiguity of the centres for the arm and the leg in the brain of the monkey causes us to assume hypothetically the existence of a similar topographical relationship for these centres in the brain of man (Fig. 42). Clinical facts lend support to this view; and the difficulty of finding uncomplicated cases of crural monoplegia seems in great part due to the tendency of an initial lesion in the leg centres to spread to, or else to cause functional perturbations in, the contiguous arm centres. The cases cited in the last section illustrate this point.

Similarly, we shall find, further on, that in many cases where a lesion seems to commence in the excito-motor centre in connection with the arm, it tends to spread either actually, or so as to cause functional disturbance in, the leg centres. We may have, however, in another set of cases, extension taking place from the arm to the face centres, rather than from the arm to the leg centres.

Thus, that we do often meet with the coincidence of paralysis of arm and leg without paralysis of face, and that we very rarely meet with paralysis of the leg and of the face without the coexistence of paralysis of the arm, as a result of a single cortical lesion, is a point which may be easily understood if we assume that the topographical relations of these centres is anything like correctly represented in the diagram (Fig. 42).

Grasset thinks that the clinical facts on record concerning unilateral convulsions warrants the following conclusion :—" When convulsions are confined to the limbs or begin in one or other limb, the lesion always exists in the upper two-thirds of the ascending convolutions (with the paracentral lobule) or in the contiguous portions of the frontal and parietal convolutions"—that is to say, around the upper two-thirds of the fissure of Rolando. This then is the diagnostic indication in cases of epileptic hemiplegia, or 'Jacksonian epilepsy,' as Charcot has proposed to term it, in which the spasm begins in either limb (Fig. 87). In the great majority of such cases we might with confidence predict the existence of some gross lesion in this situation—in other exceptional cases, however, this form of convulsion may (as there is good reason to believe) present itself without the existence of any gross lesion.

On this subject Ferrier writes:—"From the mere occurrence of an unilateral convulsion with loss of consciousness, or of an epileptic attack in which the convulsions are exhibited mainly on one side, we can form no opinion as to the nature or seat of the lesion. We may suppose, and with reason, that the opposite cerebral hemisphere is more particularly at fault; but there need be no discoverable lesion; and should a lesion exist, it need not be in any definite position. This is in accordance both with clinical facts and also with those of experiment, for I have found that long-continued irritation applied to any part of the hemisphere other than the motor area may result in an attack of unilateral convulsions. If, however, the convulsion be of the character of a monospasm, or, if, tending to become generalised, it begin invariably in the same way and does not cause loss of consciousness, and if it be followed by paresis or paralysis more or less permanent, we may diagnose an irritative lesion of the motor area of the opposite hemisphere."

(d). *Brachial Monoplegia.*—As in the case of crural monoplegia, so here, it is very important to determine in all cases, if possible, which of the movements of the upper extremity are particularly affected—that is, in cases where we have not to do with an absolute paralysis, but only with one of a partial type. Such a determination may guide us to a more minute regional diagnosis than would otherwise be possible. This may easily be understood by reference to Fig. 12, showing the position of the different centres defined by Ferrier. Nevertheless it must not be expected that a clinical diagnosis can be carried to that degree of minuteness which physiological experiment appears to render possible. The effects of morbid changes seem to diffuse themselves, so that a wider range of symptoms are often produced than might be expected from the mere lesion itself, if we look only to the visible extent of the cortex which it implicates.

This section may be best illustrated by selecting a few of the most typical of the recorded cases, bearing upon this localisation. I confine myself to some of those which have been cited by Ferrier. Others may be found in his work and in that of Grasset ('Des Localisations,' 3^{me} Éd., p. 193). The latter author also collects twelve typical cases of brachial monospasm illustrating the localisation of the arm centres in the cortex (*l. c.* p. 152). Clinical evidence in regard to this point is, therefore, much fuller and more precise than in regard to the localisation of the leg centres.

Raynaud has reported a case of paralysis limited to the left arm and more particularly to the extensor muscles, though the flexors were also paretic. There was no diminution in sensation, or alteration in electro-motor contractility. The lesion was situated in the ascending parietal convolution of the right hemisphere in the position of a line drawn back from the second frontal. It was an area of softening surrounding a tubercle the size of a millet-seed, and of about a centimètre in diameter; the softening involved the adjacent medullary fibres more than the cortex. A smaller area of softening of the size of a pea was situated near this, but within the fissure of Rolando. The whole lesion could be covered with the pulp of the finger (Fig. 46).

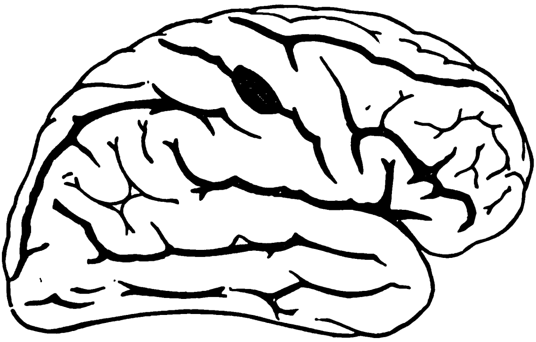

Fig. 46.

Sabourin records a case of sudden partial right hemiplegia, without loss of consciousness, which soon disappeared, leaving paralysis of the right hand and arm, which continued till death seven days afterwards. A focus of red softening was found in the left hemisphere of the size of a two-franc piece, the centre of which, where the softening was greatest, corresponded with the junction of the ascending parietal convolution and the supramarginal lobule. The softening extended half-way up the ascending

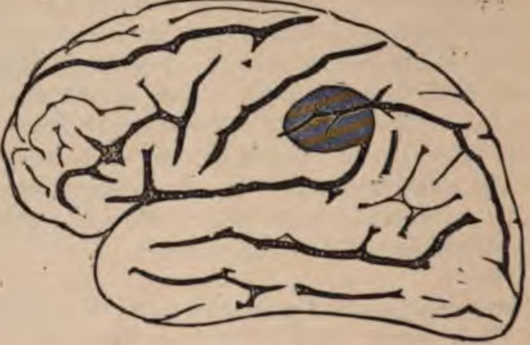

FIG. 47.

parietal convolution, somewhat concealed within the fissure. The ganglia were intact (Fig. 47).

Ringrose Atkins has recorded a case of paralysis of the right hand and arm in a general paralytic, which came on a few days before death. The membranes were adherent and the cortex was softened in a region including a small portion of the middle of the ascending frontal, the middle third of the ascending parietal convolution (extending further below than above the anterior edge of the gyrus supra-marginalis), and a little piece of the gyrus angularis, as illustrated in the accompanying figure (Fig. 48). A peculiar feature in this case, worthy of note in connection with the posterior extension of the lesion, was the presence of visual hallucinations.

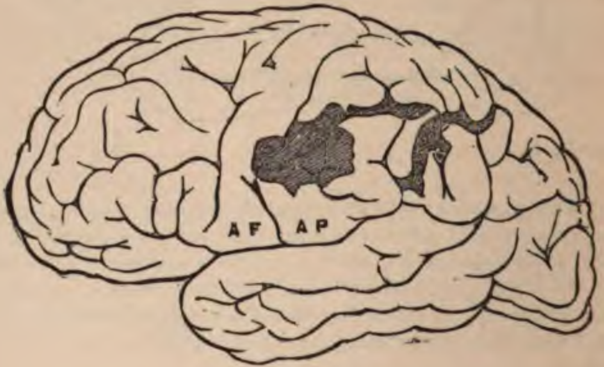

FIG. 48.

Pierret has related a case in which there was an apoplectic attack with early rigidity of the left arm and retraction of the right angle of the mouth, followed by partial paralysis of the left arm. Death occurred twelve days afterwards, from sudden coma and epileptiform convulsions. In the right hemisphere, at the junction of the middle frontal with the ascending frontal convolution, a focus of red softening of the size of a franc-piece existed. A similar focus was found in this case, in the median occipital convolution of the same hemisphere.

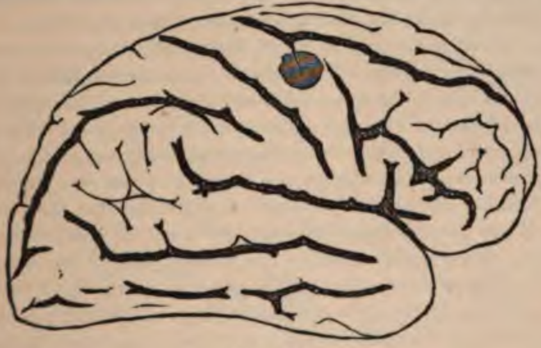

FIG. 49.

Gowers has recorded a case of congenital absence of the left hand, in which after death the middle portion of the ascending parietal convolution in the right hemisphere was found to be much smaller than the corresponding convolution in the left hemisphere.

Again, in one of my own patients an arrest of development of the left upper extremity, with practical absence of the hand, coexisted with an extremely small size of the opposite ascending parietal convolution, in the middle three-fifths of its course. The case has been recorded by Horsley and myself in 'Brain,' Vol. II.

In addition to this evidence, I will cite two of the most typical cases of brachial monospasm, one of which has been placed upon record by Hughlings Jackson (included among the twelve typical cases previously alluded to as being cited by Grasset), and the other by Dreschfield.

In H. Jackson's case, the recurring spasms were nearly always limited to the right arm, and were followed by temporary paralysis of that arm after each fit. The lesion, whose situation was diagnosed during life, was a nodule occupying the posterior extremity of the first frontal convolution where it joins the ascending frontal. It was noted that the spasms in this case always began in the shoulder, and went down the arm, contrary to the usual order. It is, therefore, worthy of note that the lesion occupied the situation of, or was immediately contiguous to, the centres which according to Ferrier are concerned with the movements of the arm as a whole rather than with those of the fingers or wrist (Fig. 12).

Dreschfield also records an extremely interesting case of brachial monospasm, dependent upon syphilitic disease, in which the situation of the lesion was likewise diagnosed during life. The patient suffered from repeated attacks of convulsion limited to the left arm, of which the phenomena were "sudden clenching of the fist, flexing of the wrist, and pronation of the fore-arm of the left side, the left angle of the mouth being at the same time strongly drawn downwards. This sudden tonic spasm lasted for several seconds, and was then followed by a few clonic spasms of the same extremity and a slight tremor of the arm; the patient at the same time was very agitated and looked very pale, but remained perfectly conscious. He stated that these paroxysms had always had the same character, varying only in degree." Death occurred from phthisis two years after the first onset of the disease. At the autopsy the dura mater was found adherent to the brain on the right side, over a space including the greater part of the ascending parietal convolution and the supra-marginal lobule. It is especially worthy of note that here we had spasms beginning in the fingers and hand, and that the lesion involved just the region in which, in the monkey, the movements of these parts have been specially localised (Fig. 12, *a*, *b*, *c*, *d*).

(e). *Brachio-Facial Monoplegia.*—This combination is one which is more frequently met with as a result of cortical disease than either brachial or facial paralysis singly.

When the paralysis is met with on the right side of the body it is very apt to be associated with aphasia, though this is not at all a necessary complication, as may be seen from the first of the two subjoined examples of this kind of paralysis.

Dieulafoy has recorded the case of a woman, aged 60, who was suddenly seized with paralysis of motion in the right arm and right lower facial region. Sensibility was unimpaired. Death occurred the day after from coma. The necropsy revealed a hæmorrhagic extravasation, the size of a nut, surrounded

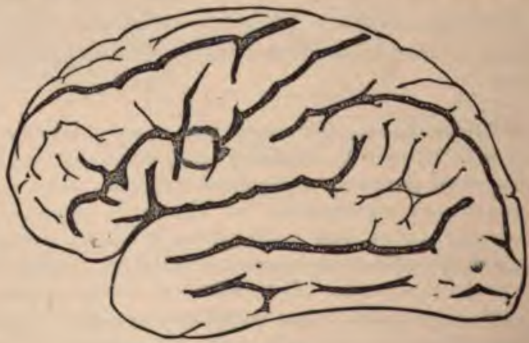

FIG. 50.

by a zone of softening in the medullary fibres of the ascending frontal convolution of the left hemisphere, posterior to the third frontal convolution (Fig. 50).

Hippolyte Martin has recorded a case of left facial paralysis, with paresis of the left arm, more particularly shown in the first three fingers. There was no aphasia. The affection had come on suddenly, without loss of consciousness or other paralysis, five or six months previously. The necropsy revealed a patch of yellow softening in the lower fifth of the ascending parietal convolution of the right hemisphere. The softening extended up the fissure of Rolando to the level of the extremity of the second frontal convolution (Fig. 51). The basal ganglia and the rest of the brain were normal.

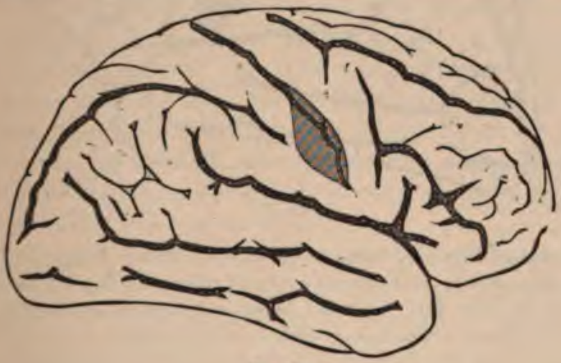

FIG. 51.

From these, and from many other very similar cases which have been recorded, it would appear, as Ferrier says (*loc. cit.*, p. 86), that "The lesions causing brachio-facial paralysis are all towards the middle or lower third of the ascending convolutions, where experiments on monkeys establish the position of the facial and manual centres." (Fig. 12, areas 7, 8, 11.)

(f). *Facial Monoplegia.*—Facial paralysis of the ordinary cerebral type rarely exists alone as a result of cortical disease. It usually occurs either associated with brachial paralysis or with aphasia. The latter association is more especially prone to occur when the lesion is on the left side; a similar lesion on the right side would be more likely to cause paralysis of the face alone, unless it happened to occur in a markedly left-handed person.

Subjoined are two of the examples cited by Ferrier.

Hitzig relates the case of a French soldier, who, two months after a bullet wound on the right side of the head, began to be affected with clonic spasms in the left side of the face. These were followed by transient, but complete, paralysis of the left side of the face and left side of the tongue. Clonic spasms occurred also in the left hand. After death, an abscess was found corresponding to the seat of injury, situated in the ascending frontal convolution, between the præcentral fissure and the fissure of Rolando (Fig. 52). It should, however, be noted that there were indications of meningeal inflammation over the whole surface of the right hemisphere, though there was no cerebral softening except in the neighbourhood of the abscess. A similar case is recorded by Wernher.

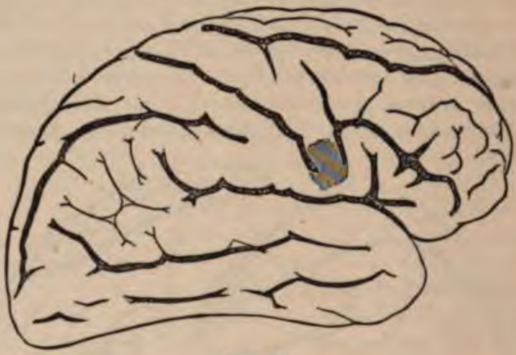

FIG 52.

With a lesion similarly situated in the left hemisphere, we may pretty confidently look for the addition of aphasic symptoms, as in the following case recorded by Hervey. Here the symptoms during life were right facial paralysis with aphasia; and, after death, a focus of softening was found anterior to the

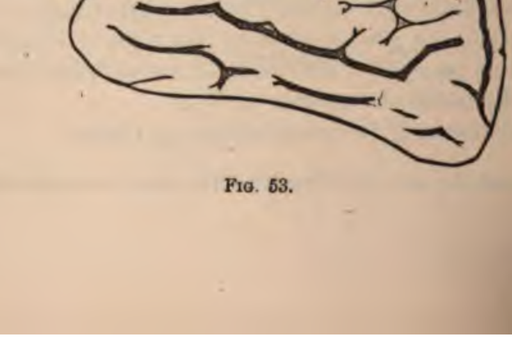

FIG. 53.

fissure of Rolando, at the junction of the third frontal with the ascending frontal convolution of the left hemisphere (Fig. 53).

Many other examples of this form of paralysis are cited by Grasset.

(g). *Aphasia.—Oro-Lingual Hemiparesis.*—We must now approach the consideration of a localisation which was the first to be attempted, with any success, in the cerebral cortex.

The successive stages by which the attempt was made have given rise to much discussion. Grasset thus sums up the evidence on this point :—" Bouillaud was the first to give a clinical confirmation of the assertion of Gall, who localised the faculty of language in the two anterior lobes of the brain; Dax first established the fact that its seat is to be found not in the two anterior lobes, but specially in that of the left cerebral hemisphere; and, finally, Broca first established the fact that it is the third frontal convolution which is the more special seat of this important centre."

The oro-lingual centres of Ferrier (Fig. 12, 9, 10) are located in the hinder part of the third frontal convolution and the contiguous lower extremity of the ascending frontal. He says :—" These centres, as I have shown experimentally in monkeys and other animals, have a more or less bilateral action. Hence, unlike the effects of destructive lesion of limb centres, destructive lesions of these centres in the one hemisphere do not cause paralysis, but only, if anything, slight unilateral weakness or hemiparesis of the oro-lingual movements. As is well known, lesions of this region in the left hemisphere are generally associated with aphasia or speechlessness. The symptoms of lesion here have both an objective and subjective aspect. The former is oro-lingual hemiparesis, the latter is the remarkable psychological affection—aphasia."

This localisation is now generally considered to be well established; reasons, however, have been already given (pp. 129 and 292) in favour of the view that any such localisation, though correct so far as it goes, is much too exclusive. In the great majority of cases, aphasia occurs in association with a partial or complete hemiplegia. In other instances, it presents itself alone, or with some very slight amount of facial paralysis.

It is interesting to note, in reference to the general question of the side of the brain involved in these cases, that Seguin found from an analysis of 266 cases of hemiplegia with aphasia, that in 243 there was right hemiplegia, whilst in only 17 was there the coexistence of left hemiplegia. It has been found, moreover, in some of the cases in which aphasia has occurred from a lesion in the right hemisphere, that the persons have been distinctly left-handed. In this way, some of the apparent exceptions only tend to confirm the general rule, that incitations to the muscular acts involved in speech pass down from one cerebral hemisphere more especially—the hemisphere which is generally potent, being that which is most frequently called into play for the execution of voluntary movements of all kinds.

One reason why aphasia sometimes exists alone, or with some slight amount of facial paralysis (generally on the right side) is to be found in the fact that the first cortical branch of the Sylvian artery (the external and inferior frontal) is small and distributed almost exclusively to the posterior part of the third frontal convolution. The blockage of this branch by embolism or thrombosis, therefore, may give rise to a localised softening with the production of permanent aphasia; while, in other cases, a spasmodic contraction of this same vessel, more or less prolonged, may possibly give rise to a temporary aphasia.

Charcot records the case of a woman who was under his care at La Salpêtrière. She had been seized with aphasia but there existed no trace of paralysis either of movement or of sensibility. In this patient aphasia was the sole morbid symptom, and atrophy of the third frontal convolution was the only lesion revealed by the autopsy—probably due to an old occlusion or narrowing of the external and inferior frontal branch of the middle cerebral artery.

Some instances of uncomplicated aphasia due to traumatic causes have also been recorded, of which two examples may be cited.

Sydney Jones describes a case of aphasia resulting from fracture of the left side of the skull by the kick of a horse. After death an abscess of the size of a nut was found in the medullary substance of the third left frontal convolution.

Simon gives the case of a healthy man who, having fallen from horseback, was found, by a physician who came up, to be aphasic, and without any other signs of paralysis. Death occurred from meningitis. A small wound with depressed fracture of the left side of the skull was found; and corresponding to this internally there was a cerebral softening—surrounded by meningeal inflammation—involving the third left frontal convolution (in which a spiculum of bone was imbedded), the second frontal, and the convolutions of the island of Reil.

A most remarkable case of double symmetrical lesion of the third frontal convolution has been recorded by Barlow ('Brit. Med. Jrnl.,' July 28, 1877), which is as yet unique.

A boy aged 10, the subject of aortic disease, of which he ultimately died, was seized with right hemiplegia, chiefly brachio-facial, and aphasia. From this he had apparently recovered at the end of a month. Three months after, he was seized with left brachio-facial monoplegia. This time there was not only aphasia, but paralysis of all voluntary movements of the face and tongue. Reflex deglutition, however, was unimpaired. There was no affection of sensation in the paralysed parts, either in the skin or mucous membranes of the palate, etc., and the muscles reacted normally to the electric current. There appeared to be loss of voluntary motor power over the muscles concerned in deglutition and articulation. This lasted till death, though the arms improved somewhat in power. Intelligence was fair and comprehension good. On post-mortem examination, a lesion was found in each hemisphere, in exactly corresponding situations. The region involved by each lesion—which was yellow softening —was the lower end of the ascending frontal, and the hinder end of the middle and inferior frontal convolutions (Fig. 54).

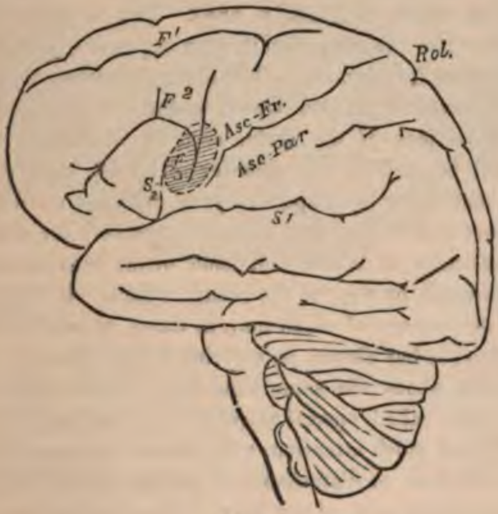

Fig 54.

In its first stage this was a typical case of aphasia and partial right hemiplegia; after the onset of the second lesion a complete aphasic condition became established, and in this stage the case—apart from its previous history—closely resembled one of bulbar paralysis.

There is now no reasonable room for doubt that a lesion occupying the hinder part of the third frontal convolution in one cerebral hemisphere—and by far the most frequently in that of the left side—gives rise to the common kind of aphasia.

There is also some reason to believe that aphasia associated with 'word-deafness' may be caused by a lesion of the upper temporal convolution (in the situation of Ferrier's 'auditory centre') near the posterior extremity of the Sylvian fissure (see p. 122).

It seems certain, *à priori*, that the auditory centre must be connected by means of commissural fibres with the centres situated in the posterior part of the third frontal convolution (whether we call them motor or whether we regard them as kinæsthetic centres); and it seems equally clear that a lesion which should cut across these commissural fibres in any part of their course between the posterior extremity of the Sylvian fissure and the third frontal convolution ought also to produce a typical aphasic condition (Fig. 7). This conclusion I announced in 1880 ('Brain as an Organ of Mind,' p. 686). This *à priori* deduction is not without evidence in support of it, even at present, though more will doubtless soon be forthcoming. The fibres connecting these two parts of the cortex have not yet been traced by the anatomist. If, however, we examine sections of the brain, such as are shown in Figs. 55 and 60 it will be seen to be a matter of almost complete certainty that fibres connecting the upper temporal with the third frontal convolution must pass in the first place (*a*) not very far away from the posterior extremity or sensory division of the internal capsule, and thence (*b*) onwards by way of the island of Reil. But these are just the regions the damage of which may, at times, as much clinico-pathological experience has shown, be associated with aphasia.

(a). Grasset, about the same time that I dwelt upon the above-mentioned view, called special attention to a fact which had not previously attracted much attention, viz., the not unfrequent association of aphasia with loss or disturbance of general sensibility (hemianæsthesia) on the right side of the body ('Des localisations,' 1880, 3^me^ édit., pp. 272-277). He refers to several cases illustrating this association, but gives, as I venture to think, an erroneous explanation when he attributes it to the supposed propinquity of the third frontal convolution and the sensory segment of the internal capsule (*loc. cit.*, p. 277). A reference to Fig. 55 will show that the posterior part of the hinder segment of the internal capsule is far removed from the region of Broca and the contiguous portion of the insula, though it

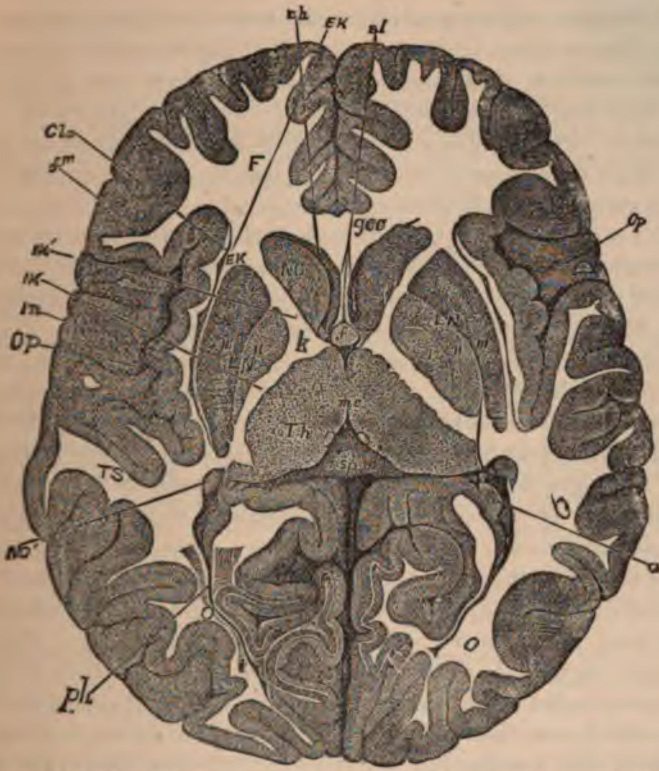

FIG. 55. HORIZONTAL SECTION OF THE BRAIN OF A CHILD NINE MONTHS OLD, THE RIGHT SIDE BEING AT A SOMEWHAT LOWER LEVEL THAN THE LEFT HALF [after Flechsig].*

F, frontal; TS, temporo-sphenoidal; and O, occipital lobes. Op, operculum. In, Island of Reil. Cls, claustrum. f''' third frontal convolution. Th, thalamus, NC, caudate nucleus. NC', tail of caudate nucleus. LN, lenticular nucleus. 1,11, 111, first, second, and third divisions of the lenticular nucleus. EK, external capsule. IK, posterior division; IK', anterior division; K, knee of the internal capsule. ah, ph, anterior and posterior horns of the lateral ventricles. gcc, knee of the corpus callosum. sp, splenium. mc, middle commissure. f, fornix. sl, septum lucidum. a, cornu Ammonis.

must be very close to the posterior part of the commissural fibres connecting the upper temporal gyrus with Broca's region.

(b). Again, the island of Reil lies in the direct track which must almost certainly be taken by such commissural fibres. But, since 1868 when Meynert originally advanced the notion, and supported it by cases, that a lesion in the island of Reil might produce a typical aphasic condition, other cases of the same kind have been published, and these have been analysed by Boyer ('Études topographiques sur les lésions corticales,' Thèse de Paris, 1879, N°. 115). There are now over thirty of such cases on record,* so that there can be no doubt as to the correctness of Meynert's position.

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* To obtain this view, Flechsig makes a horizontal section of the brain just above and parallel with the Sylvian fissure.

As I said therefore in 1880, Broca's special localisation of the third left frontal convolution as the seat of aphasia-producing lesions, must be held to hold good only for one particular, though very common, form of Aphasia, and not exclusively even for this form.

The above considerations may make us thoroughly admit the real validity of the objections raised by some against the old doctrine of Broca and his immediate followers, that the posterior part of the left third frontal gyrus is *the* region always damaged in cases of Aphasia. We must now be prepared to admit the existence of many closely allied forms of Aphasia and a comparatively wide area in which lesions may give rise to this or that variety. Subsequent experience tends to confirm the hypothesis which I then advanced, to the effect that the tendency to mental impairment with Aphasia, and the degree of such impairment, will, other things equal, increase as lesions of the left hemisphere, capable of producing this condition, recede in site from the third frontal convolution, and approach the posterior extremity of the Sylvian fissure.

PARALYSES DUE TO LESIONS OF THE CENTRUM OVALE.

Pitres has endeavoured to systematise our knowledge concerning the effects of lesions in the centrum ovale. Under this term he includes the whole of the medullary substance intervening between the cortex and the basal ganglia.

With a view to the more accurate recording in future of the precise situation and extent of lesions in this part of the brain, he makes a series of vertical sections of the cerebral hemisphere at right angles to its longitudinal axis.

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* There seems some reason also for supposing that an ordinary aphasic condition may be produced by a lesion which cuts across the efferent fibres proceeding from the third frontal convolution on the potential side. Two examples of this type will be found cited in the next section (p. 296). On the whole, however, I am strongly disposed to think that such cases would prove to be of aphemic type rather than true cases of aphasia (See p. 119).

The præ-frontal section is carried through the præ-frontal lobe.

The next section made, two centimètres in advance of the fissure of Rolando, through the bases of the three frontal convolutions, is named the pediculo-frontal section (in which are to be distinguished three fasciculi, viz., the superior, middle, and inferior frontal fasciculi, corresponding, externally, with the three frontal convolutions).

Then comes the frontal section, which is made by dividing the hemisphere through the ascending frontal convolution, but parallel to the fissure of Rolando (containing also superior, middle, and inferior frontal fasciculi).

The parietal section is made in a similar manner, but is carried through the ascending parietal convolution. In it may be recognized superior, middle, and inferior parietal fasciculi.

Next comes the pediculo-parietal section, which is made by cutting through the hemisphere parallel to the fissure of Rolando, but three centimètres behind it. This passes through the superior and inferior parietal lobules, and is composed of superior and inferior parietal fasciculi.

Lastly, a section through the middle of the occipital lobe gives the occipital section.

Facts of importance concerning the Vascular Supply of the Centrum Ovale.

Part of the vascular supply of the centrum ovale is derived from the cortical, and part from the basal system of vessels. According to Duret the two sets of vessels do not anastomose with one another. The part of the white substance which is supplied by the cortical system derives its supply from what are known as the long nutrient arteries (Fig. 2), and these, according to Duret, penetrate into the medullary substance for a depth of 3-4 centimètres (or about 1¼ to 1⅝ of an inch). The centrum ovale receives, therefore, only very small vessels. It is thus no matter for surprise that only very small hæmorrhages (size of a pea to a small almond) should occur within its substance.

Large hæmorrhages, of course, often extend into the centrum ovale from the internal capsule and its neighbourhood.

Summing up the results so far obtained by Pitres, Ferrier writes ('Localisation,' p. 52) :—"Now it is only in certain of the medullary fasciculi so marked out, that lesions cause paralysis of motion and degeneration of the motor tracts. These regions are included in the pediculo-frontal, frontal, and parietal sections, or generally, in the fronto-parietal area. Lesions here have exactly the same effect as lesions of the corresponding cortical region, according as they are destructive or irritative, or according as they are limited or general. And M. Pitres has brought forward evidence of a very satisfactory kind to show that the early rigidity and muscular spasms which so frequently accompany hemiplegia with effusion into the lateral ventricles, are essentially due to irritation of the fronto-parietal fasciculi of the centrum ovale."

Two or three typical cases may be cited showing the effects of lesions in the fronto-parietal area of the centrum ovale in producing complete or incomplete hemiplegia, with or without aphasia.

Hodgson has related a case of right hemiplegia with aphasia, followed several months after the seizure by late rigidity of the right arm. Death occurred from chronic bronchitis more than a year after the attack. In the centrum ovale of the left hemisphere, was a cavity nearly empty, one inch and a quarter long, situated external to the lateral ventricle and between its anterior horn and the island of Reil. The rest of the brain, except the part immediately around the cavity (which was yellowish), presented nothing abnormal.

Again, in a case observed by Pitres himself of right hemiplegia and aphasia, no lesion was found in the cortex in Broca's region, but on section a zone of softening was found in the centrum ovale, affecting the inferior pediculo-frontal fasciculus (Fig. 56).

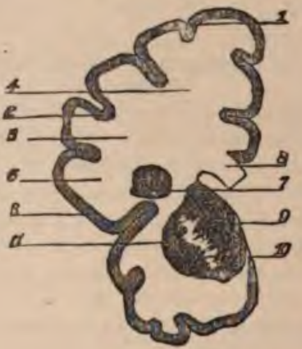

FIG. 56.

Anton Frey has recorded a case in which during life the symptoms were paresis of the left arm and left side of the face, without any affection of sensibility. Death occurred from gangrenous erysipelas of the face. In the right hemisphere a small focus of hæmorrhage formed by the juxtaposition of three minute extravasations, each of the size of a mustard seed, was found in the medullary fibres at the junction of the middle frontal with the ascending frontal convolution (Fig. 57). Thus it would appear that a brachio-facial monoplegia may be produced by lesions limited to the medullary fibres of the

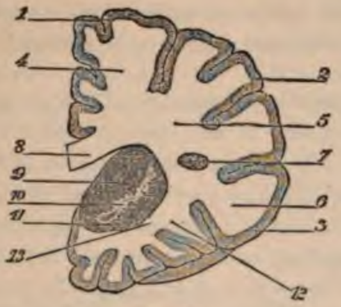

FIG. 57.

middle fasciculi of the pediculo-frontal and frontal sections.

Pitres records another case in which a brachio-crural monoplegia had existed during life, and where, after death, no lesion of the cortex was found, but instead

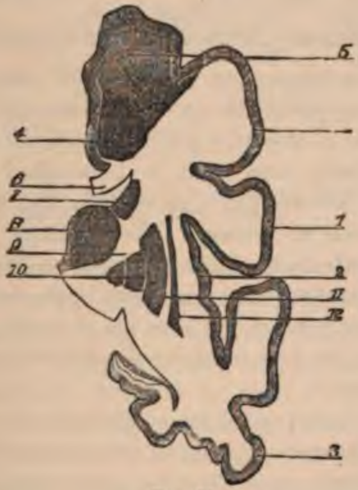

FIG. 58.

a lesion situated in the medullary fasciculi of the superior frontal and parietal region (Fig. 58).

It should be remarked, however, in connection with paralyses produced by lesions in this part of the centrum ovale, that they are attended by no specific characters by which they can be separated, during life, from the results of cortical lesions on the one hand, or of small lesions in the internal capsule on the other.

Lesions in the medullary substance of the præ-frontal lobes are not known to give rise to any definite motor or other symptoms. Again, lesions of the medullary substance in the immediate neighbourhood of the pediculo-parietal and of the occipital sections give rise to no motor symptoms; though there may be the production of well marked sensory or mental defects—especially when the lesion occupies some portion of the middle third of the pediculo-parietal section in the left hemisphere. It seems highly probable that aphasic and amnesic defects may thus be caused; though I am not able to cite any cases in which small lesions limited to the medullary substance of the hemisphere in this situation have been observed during life and accurately recorded. There are gaps in our knowledge here which much require to be filled up by future observers.

PARALYSES DUE TO LESIONS OF THE CORPUS CALLOSUM.

Little is known concerning the signs of disease in the corpus callosum. This is partly due to the circumstance that perhaps an insufficient amount of attention has been bestowed upon the subject; though it is probably even more to be ascribed to the fact that isolated lesions are not prone to occur in this brain region, notwithstanding its size and extent. Hæmorrhages and abscesses are practically not to be looked for in its substance; softenings and tumours are the lesions most likely to be met with in the corpus callosum, but in these cases other parts of the brain are generally involved more or less simultaneously, and consequently the clinical picture is apt to be varied and indistinct.

In regard to softening of the corpus callosum we may expect to learn more in the future. It must be remembered that this structure derives its chief blood supply from the anterior cerebral artery, so that, in cases of thrombosis or embolism involving this vessel, a softening of the corresponding half of the corpus callosum (more or less complete) should be met with as one of the pathological results, and ought, therefore, to entail mental defects of a well-marked order. Owing to the nature of the functions with which it is concerned, unilateral lesions of the corpus callosum, on either side, would be capable of completely annulling its functions. The uncomplicated picture would only be obtained, however, by the occurrence of an occlusion limited to the vessel which supplies this structure, viz., the middle internal branch of the anterior cerebral (Fig. 59, II)—an event only to be looked for with great rarity.

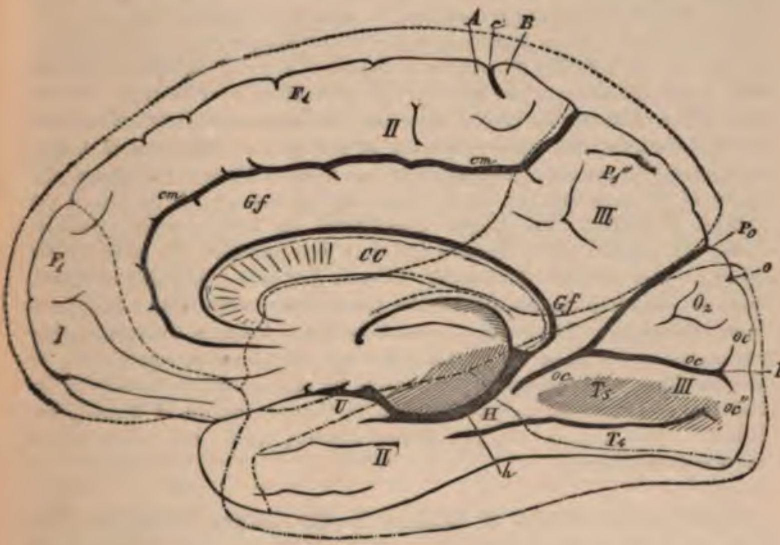

FIG. 59. INNER SURFACE OF THE RIGHT HEMISPHERE [after Ecker], SHOWING DISTRIBUTION OF CORTICAL VESSELS [after Duret].

The sudden cutting off, in an adult, of the functional activity of such a part as the corpus callosum, might well be supposed to produce marked symptoms—even though well defined defects may have been absent in some of the recorded cases in which there has been a congenital deficiency (in whole or in part) of the corpus callosum. These two sets of cases stand on a different level.

It should be borne in mind, therefore, that a thrombosis of the anterior cerebral artery, when it involves the vessel beyond the anterior communicating artery, ought to render the corpus callosum functionally inert and, consequently, to be attended with well-marked mental defects.

Again, in cases where thrombosis simultaneously affects the left anterior and middle cerebral arteries (perhaps beginning in the common carotid), we might expect to find right hemiplegia and aphasia associated with an altogether unusual amount of mental degradation, in addition to blindness and loss of smell on the side of the lesion.

Elsewhere ('Brain as an Organ of Mind,' p. 484) I have said:—"These transverse 'commissural' fibres are of much interest, because there is reason to believe that they are, to a considerable extent, in relation with that unification of Consciousness which unquestionably exists (as everyone can testify) in spite of the fact that the organs of Sensorial Activity are double throughout. Such commissures are also, in all probability, very essential for the carrying on of the higher mental processes. In cases recorded by Dr. Langdon Down and others, the non-development of this part of the Brain in human beings has been associated with more or less marked Idiotcy; but then, the arrest of development has for the most part not been strictly limited to the 'Corpus Callosum.' The Middle Commissure, the Fornix, or some convolutional regions, have been often at the same time deficient. In some of the recorded cases in which the Corpus Callosum has been only partially absent, there has been less degradation of the Intellectual Powers than might have been anticipated. In certain of these latter cases, however, the persons have either died so young, or the morbid conditions have been so complicated, as to make them of comparatively little value for settling the question as to the real importance of the Corpus Callosum in the carrying on of mental processes." (See Knox in 'Glasgow Medical Journal,' April, 1875, where fifteen cases are referred to.)

Signs and Symptoms of Lesions in the Corpus Callosum.—The only facts which I can adduce on this subject are some which have recently been published by Bristowe ('Brain,' Oct., 1884), based upon three cases of tumour in the corpus callosum. In each of these cases the tumour grew in an unequal fashion into both cerebral hemispheres, and Bristowe expresses the opinion that some of the most striking symptoms were caused by this extension of the lesion from its original site. Still, if such a mode of growth is to be looked upon as a common tendency of tumours in this situation—as seems to be not improbable—we must regard the symptoms referred to as an essential part of the picture by which we are enabled (if at all) to recognize the presence of a lesion in the corpus callosum.

According to Bristowe, the chief characteristic features of his cases were:—"1st, their ingravescent character, a character which they possessed in common with other cases of cerebral tumour; 2nd, the gradual coming on of hemiplegia, for the most part resembling in its distribution the paralytic symptoms usually attending hæmorrhage into one of the hemispheres, or softening due to embolism; 3rd, the association with the paralysis of one side, of vague hemiplegic symptoms of the other; 4th, the supervention of stupidity, associated for the most part with extreme drowsiness, a puzzled enquiring look when awake, a difficulty of getting food down the throat, and cessation of speech—I say a difficulty of getting the food out of the mouth rather than paralysis of the mouth and throat, and I say a cessation of speech rather than aphasia or loss of articulating power, because it seemed to me, in watching the cases, that these phenomena were due mainly to stupidity and irresistible tendency to sleep, and not definitely to paralysis or affection of the centres for speech; 5th, the absence of implication of the oculo-motor nerves, and of direct implication of other cerebral nerves; and lastly, death from coma. It is further noteworthy that some of the more striking symptoms of cerebral tumour were either absent or only slightly developed. Headache did not appear to be very severe in any case, and in one is not so much as mentioned; there was practically no sickness; optic neuritis was certainly absent, at any rate in one case, up to within a week or ten days of death; from first to last the patients were free from epileptic attacks, and never had anything that could be termed a fit."

In these cases recorded by Bristowe, no distinct symptoms were noted till within 10-14 weeks from the date of death. He points out, however that in each case the total bulk of the tumour was very large, "and must have taken much longer to grow than the clinical history gave as the duration of the disease." He adds:—"In all of them the median portion of the corpus callosum was evidently the starting point of the disease, and this body must have been largely involved before the patient complained of definite symptoms."

The symptoms above referred to Bristowe believes to be due chiefly to the extension of the growth into the two cerebral hemispheres, for the most part equally (or with unequal rates), as well as to the "diffused pressure on important parts caused by the great collective bulk of the tumour," such pressure being evidenced by the extreme flattening of the surface of the hemispheres and the singular absence of both subarachnoid and ventricular fluid. It is certainly true that many of the symptoms met with in Bristowe's cases are explicable by reason of the implication of both cerebral hemispheres in the disease; and, therefore, it is here again extremely difficult to define how much of the mental impairment is to be attributed to the loss of function in the corpus callosum itself, and how much to the mere fact of the existence of disease in both hemispheres, coupled with a great increase of intra-cranial pressure. This, however, is a matter principally of physiological importance, and does not so much concern us from our special point of view, which should be that of the clinical observer. In all probability, had these patients come under skilled observation in the early stages of their disease, we should have known something more definite as to the premonitory symptoms—that is, those dependent upon the growth of the tumour in the substance of the corpus callosum itself.

We have only to look to the kind of symptoms by which a tumour originating in the corpus callosum is likely to be attended during its progress towards a fatal issue, in order to see whether they constitute a group having fairly distinctive characters—no matter how such symptoms are produced, so long as they are likely, in subsequent cases, to be evolved in something like the same order. The cases already recorded have, unquestionably, presented very similar characters ; and these have been of such a kind that, having due regard to their slow and gradual onset, they ought to aid us in arriving at a correct diagnosis in future cases running anything like a similar course.

Briefly then, according to Bristowe, the order of events may be expected to be something like this:—

"First the occurrence of headache, and other somewhat vague symptoms of progressive cerebral disease. Second, the gradual onset of more or less well marked hemiplegia. Third, the appearance in a greater or less degree of similar symptoms on the opposite side of the body. Fourth, the coming on of dementia, with drowsiness, loss of speech, difficulty in swallowing, and want of control over the rectum and bladder."

II.—LESIONS IN PARTS SUPPLIED BY THE BASAL ARTERIAL SYSTEM.

DISTRIBUTION OF VESSELS PERTAINING TO THE BASAL ARTERIAL SYSTEM.

1. The Middle Cerebral or Sylvian Arteries. The basal branches of the Sylvian arteries are much more numerous than those of the anterior or of the posterior cerebral arteries. They spring from its upper border, enter the orifices of the perforated space, and supply nearly the whole of the corpus striatum, as well as the internal capsule and part of the optic thalamus.

These basal branches are given oft near the origin of the vessel (Fig. 1, 3, 3). They vary from ½-1½mm. in diameter. They pass for a short distance over the outer and under surface of the lenticular nucleus (where they present a fan-like arrangement ; thence into its substance, and through it to the internal capsule (Fig. 60). In front, some of these vessels reach and in part supply the anterior portion of the caudate nucleus; while the more posterior branches, after passing through and supplying twigs to that portion of the internal capsule, reach and nourish the outer and anterior part of the thalamus.

These vessels are divided by Duret into two groups, viz. (1) internal branches, a set of small branches which penetrate the two inner segments

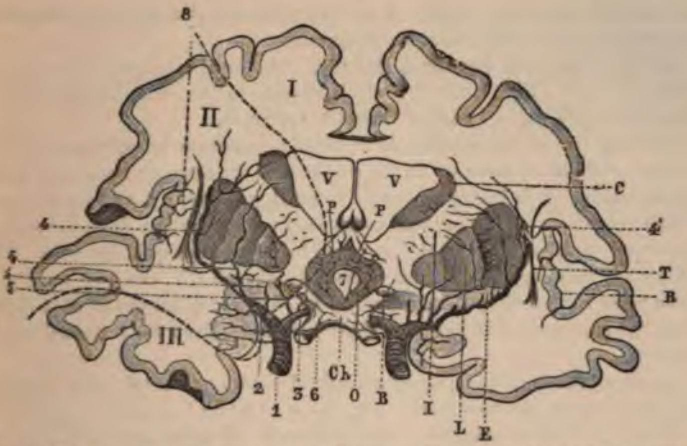

FIG. 60. TRANSVERSE SECTION OF THE CEREBRAL HEMISPHERES MADE ONE CENTIMETRE BEHIND THE OPTIC CHIASMA, SHOWING THE ARTERIES OF THE CORPUS STRIATUM [after Duret].

ch, Optic Chiasma. B, Section of Optic tract. L, Lenticular nucleus. I, Internal Capsule or foot of Reil's corona radiata. C, Caudate or intra-ventricular nucleus of the Corpus Striatum. E, External Capsule. T, Claustrum. R, Convolutions of the island of Reil. V, V, Section of the Lateral Ventricles. P, P, Pillars of the Fornix. O, Grey Substance of the Third Ventricle continuous posteriorly with the Optic Thalamus.

Distribution of Vessels.

I. Is the territory of the *Anterior Cerebral Artery.*
II. " " " *Sylvian Artery.*
III. " " " *Posterior Cerebral Artery.*

1, internal carotid artery. 2, Sylvian artery. 3, anterior cerebral artery. 4, 4, external arteries of the corpus striatum (lenticulo-striate arteries). 5,5, internal arteries of the corpus striatum (lenticular arteries).

The opto-striate arteries are not represented.

of the lenticular nucleus and contiguous parts of the internal capsule; and (2) the larger external branches, which are again divisible into two sets—such as have been referred to above—that is, an anterior set named lenticulo-striate arteries; and a posterior set, the lenticulo-optic arteries. One vessel of the former set is generally of distinctly larger size than the others. It bends round or through the peripheral layers of the outer segment of the lenticular nucleus, crosses the internal capsule, and penetrates the anterior and outer part of the caudate nucleus, where it breaks up into three or four small branches. As Charcot points out, rupture of some part of this vessel occurs with extreme frequency as a cause of cerebral hæmorrhage. The effused blood is then situated either in the outer layers of or just outside the lenticular nucleus.

2. The Anterior Cerebral Arteries. The basal branches of the anterior cerebral are less constant in their distribution (Fig. 1, 1). They often furnish twigs to the head of the caudate nucleus. When they are absent their place is taken by offsets from the choroid branch of the posterior cerebral artery.

Small filaments belonging to this set also come off from the anterior communicating artery, and supply, on each side, the anterior and inner parts of the thalamus, together with the soft commissure.

3. The Posterior Cerebral Artery. The basal branches of the posterior cerebral artery (Fig. 1, 2, 4, 4) are distributed to the crura cerebri, the corpora quadrigemina, and part of the optic thalamus.

The first set of these basal twigs mostly penetrate at once into the crura cerebri; but one of them of larger size goes to the inner and posterior part of the thalamus. It is known as the internal posterior optic artery.

The second set of these basal twigs is given off further from the origin of the vessel on each side, and they proceed to the corpora quadrigemina. These bodies are well supplied by three arteries on each side, the two anterior of them being furnished by the posterior cerebral, while the third is derived from the superior cerebellar (and, therefore, comes from the vertebral arterial system).

Another branch of the posterior cerebral artery, given off after this vessel has bent round the crus cerebri, is the external posterior optic artery. It often ascends obliquely through the substance of the crus and penetrates the contiguous posterior part of the thalamus.

PARALYSES DUE TO LESIONS OF THE INTERNAL CAPSULE, THE CORPUS STRIATUM AND THE THALAMUS.

The internal capsule is an all-important band of white fibres which is, in part, a continuation upwards of a portion of the crus cerebri,

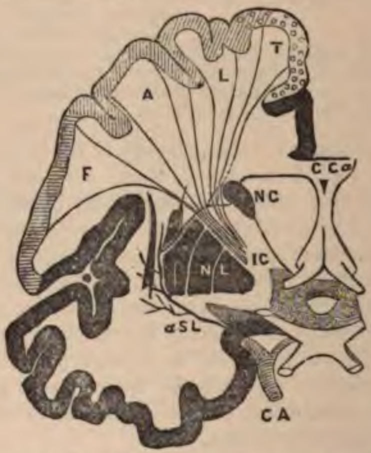

FIG. 61. SECTION OF THE LEFT CEREBRAL HEMISPHERE NEARLY IN THE PLANE INDICATED IN FIG. 42 [after Horsley].

This figure shows the relative position of the centres in the excito-motor area, and the supposed direction of fibres passing from them on their way through the Centrum Ovale to the Internal Capsule (IC).

CCa, Corpus Callosum. NC, Caudate nucleus. NL, Lenticular nucleus. CA, Internal Carotid Artery. aSL, Lenticulo-striate artery.

through the basal ganglia, into the white substance (centrum ovale) of the cerebral hemisphere. Above the level of the corpus striatum and thalamus, these same fibres diverge in fan-like fashion towards different parts of the cortex, constituting the so-called corona radiata. The internal capsule is, therefore, sometimes spoken of as 'the foot of the corona radiata.'

A portion of the fibres passing downwards, so as to constitute part of the internal capsule, viz., those from the Rolandic area of the cortex (lesions of which cause hemiplegia, or monoplegiæ of different kinds, with or without aphasia) have already received our attention in their course through the centrum ovale.

We have now to follow these same fibres, that is, to speak of the lesions which affect them in their course through the internal capsule itself, and also to consider the effect of lesions upon certain other constituents of this important aggregation of fibres.

In regard to the structural relations existing between the crus cerebri and the internal capsule, the following facts should be borne in mind.

The internal capsule is the prolongation not of the whole of the crus cerebri, but only of the foot (crusta) or lower layer. The tegmentum, or upper layer, which is separated from the foot by the locus niger (Fig. 22), enters into special connection with the corpora quadrigemina and optic thalamus, but takes no share in the formation of the internal capsule.

The internal capsule is now generally believed to be composed of (1) direct peduncular fibres which pass downwards, from the convolutions in the fronto-parietal region through the capsule, without being arrested in the ganglia; and (2) indirect peduncular fibres. Of these latter some are supposed to proceed downwards from the under surface of the caudate nucleus; others, downwards from the lenticular nucleus. They seem to emerge most plentifully from the first segment of this nucleus, but to be less and less numerous from the second and third segments. As Charcot suggests, this difference in the number of issuing fibres may account for the difference in colour of the three segments of the lenticular nucleus.

Concerning the relative proportion existing between the 'direct' and the 'indirect peduncular fibres,' there has always been much difference of opinion. Some remarks upon this subject have already been made (p. 212).

Above the internal capsule, four kinds of fibres seem to enter into the composition of the corona radiata, which is its upward prolongation. Three of these sets of fibres connect the grey nuclei with the cortex and are known as 'radiating fibres.' We have, therefore, (1) radiating fibres of the caudate nucleus; (2) radiating fibres of the lenticular nucleus (which principally proceed from the upper border of the second and third segments); (3) radiating fibres of the thalamus; (4) the direct fibres which proceed from the cortex to the foot of the crus, without being arrested in the central grey nuclei. The order of these latter fibres from before backwards has been set forth on p. 210. The hindermost section of the corona radiata is composed of sensory fibres.

We are principally concerned here with the 'direct peduncular fibres,' and something more specific will be said concerning them after a brief reference has been made to the topographical relations of the internal capsule as a whole.

Horizontal and vertical transverse sections of the brain such as are represented in Figs. 55 and 61, will show at once how very intimate are the structural relations existing between the internal capsule, and the three basal ganglia to which reference has been made, viz., the caudate nucleus, the lenticular nucleus, and the thalamus. A reference to Fig. 55, especially, reveals the important fact that the internal capsule is divisible into an anterior and a posterior segment inclined towards one another at nearly a right angle. The anterior segment lies between the caudate and the lenticular nucleus; while the posterior segment is situated between the thalamus and the lenticular nucleus. The part at which the two segments unite is commonly known as the knee (or genu) of the internal capsule.

The functions of the parts of the internal capsule made up of 'direct peduncular fibres' have already been discussed. It has been shown, for instance, that the fibres in the posterior third of the hinder segment are almost exclusively concerned with the transmission of sensory impressions, and that lesions involving this part of the internal capsule give rise to complete hemianæsthesia of the opposite side of the body (p. 152). Again, it has been shown that the fibres in the remaining two-thirds of this hinder segment of the internal capsule are concerned with the transmission of motor impressions to the limbs and probably to the trunk muscles; that those of the knee or genu are concerned with the transmission of motor incitations to the muscles of the tongue, lips, etc.; and, finally, that those in the anterior segment are concerned with the transmission of out-going impressions whose nature or purpose is unknown, but which seem, according to Brissaud, to be in relation with some kinds of mental activity (p. 204). When these latter fibres, as well as those which descend by way of the genu, are cut across by lesions, bands of 'secondary degeneration' are produced which can be traced only as far as the pons Varolii, and the bulb; while the fibres composing the anterior two-thirds of the hinder segment of the internal capsule, when similarly cut across by lesions, give rise to well-marked 'secondary degenerations' that can be traced downwards through the peduncle, pons, and medulla, into the lateral column of the opposite half of the spinal cord, as well as into the anterior column on the side of lesion.

Facts of importance concerning the Vascular Supply of the Internal Capsule, the Corpus Striatum, and the Thalamus.— The vascular supply and the pathology of the internal capsule, the corpus striatum, and the thalamus, are so closely related as to make it essential that they should be considered together.

The entire system of basal arteries springing from the Sylvian may be obstructed in consequence of thrombosis or embolism of this arterial trunk itself. Softening then attacks the internal capsule, and the mass of grey nuclei nearly in its entirety—the regions corresponding to the distribution of the anterior cerebral and the posterior optic arteries being alone unaffected. In such a case we ought to meet with the common type of cerebral hemiplegia, together with cerebral hemianæsthesia; and, owing to the necessary simultaneous involvement of the cortical system of the Sylvian, we should have, in addition, when the artery of the left side is involved, the production of aphasia together with sensory amnesia (owing to the absence of blood-supply from the territories of the first and fourth cortical branches of the Sylvian) (Fig. 28). With a similar lesion on the right side, there would probably be the absence of aphasia or amnesia, and the existence merely of some dulness, slowness of apprehension, and perhaps confusion of mind, for a time.

Where, from any cause, softening is limited to the sphere of the lenticulo-striate arteries, we may expect the production of complete motor hemiplegia, without sensory defect; but with the addition of simple aphasia or aphemia when the lesion is on the left side.

Where, again, softening happens to be limited to the sphere of the opto-striate arteries, we may expect the production of a less complete motor hemiplegia, combined with cerebral hemianæsthesia (or even with this latter condition alone), but without the complication of aphasia even when the lesion is on the left side.

Softening of the thalamus is found to be distinctly less frequent than softening of the corpus striatum.

The same kind of difference in symptomatology which is above indicated, as obtaining between softening when it occurs in the domain of the lenticulo-striate and in that of the lenticulo-optic arteries, also holds good for rather large hæmorrhages in these respective situations.

"Most frequently, contrary to the generally accepted opinion, extravasation of blood in such a case, as M. Gendrin had long truly recognized, takes place in the first instance not into the substance of the corpus striatum, but outside it and, more precisely, in contact with the external surface of the lenticular nucleus, between this surface and the external capsule, which becomes as it were detached. . . . In this manner flattened out focal lesions are produced, which in transverse sections have the appearance of straight, linear lacunæ, nearly vertical in direction and parallel to the grey nucleus of the claustrum" (Fig. 62).

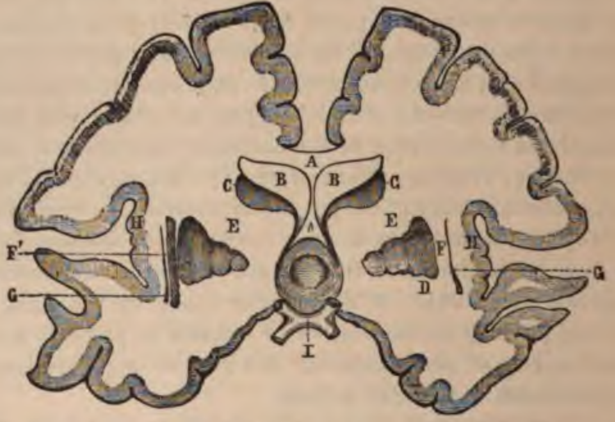

FIG. 62. EXTRA-LENTICULAR HÆMORRHAGIC FOCUS IN THE ANTERIOR PART OF THE EXTERNAL CAPSULE—TERRITORY OF THE LENTICULO-STRIATE ARTERIES [after Charcot].

(Production of incomplete Hemiplegia, without hemianæsthesia.)

A, Corpus Callosum. B, B, Lateral ventricles. C, C, Caudate nuclei. D, Lenticular nucleus. E, E, Anterior or lenticulo-striate region of the internal capsule. F, External capsule. F′, Hæmorrhagic focus destroying the external capsule. G, G, Claustrum. H, H, Island of Reil. L, Optic chiasma.

With a slight lesion of the kind represented in the foregoing figure the hemiplegia might be expected to be incomplete and merely temporary.

Where a large extravasation occurs in the same situation it will produce a complete motor hemiplegia, of a much more durable type; in the main owing to the compression exercised by the extravasated blood upon the fibres of the internal capsule. This subject has been well explained by Charcot. He points out that the extravasation tends to increase especially in a transverse direction, and owing to the greater resistance of the cranial walls on the side of the island of Reil, this (together with the claustrum and the external capsule) resists the pressure of the extravasated blood, whilst the grey nuclei are displaced, as a whole, towards the ventricular cavities (Fig. 63). It is clear that

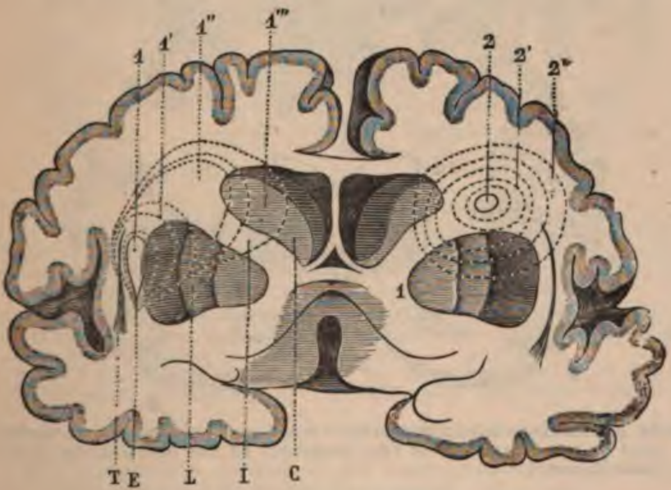

FIG. 63. DIAGRAM ILLUSTRATING SEATS AND MODES OF EXTENSION OF HÆMORRHAGES CORRESPONDING TO THE ANTERIOR PART OF THE INTERNAL CAPSULE, PRODUCED BY RUPTURE OF DIFFERENT LENTICULO-STRIATE ARTERIES [after Charcot].

(Production of Hemiplegia.)

C, Caudate nucleus. L, Lenticular nucleus of the Corpus Striatum. I, Internal capsule; E, External capsule. T, Claustrum.

1, Primary hæmorrhagic focus in the anterior part of the external capsule.

1', 1'', 1''', Progressive extension of the primary lesion (compression or destruction of the internal capsule).

2, Primary focal lesion in the anterior part of the internal capsule.

2', 2'', 2''', Progressive extension of this lesion (destruction of the internal capsule, displacement or destruction of the caudate nucleus).

the elements of the internal capsule will necessarily be more or less strongly compressed in consequence of an alteration of this nature, and thus effects may result, not notably different from what may be produced in another case by a large lesion, in the substance of the anterior part of the internal capsule itself (Fig. 63, 2-2‴). In a hæmorrhage of this kind a large quantity of blood may also extend upwards into the centrum ovale.

If now we take the case of a hæmorrhage occurring near or in the posterior extremity of the internal capsule, the effects may be quite different. Thus, where the lesion is small and where it involves quite the posterior extremity of the internal capsule (Fig. 64), the result will be the production of cerebral hemianæsthesia of a permanent type.

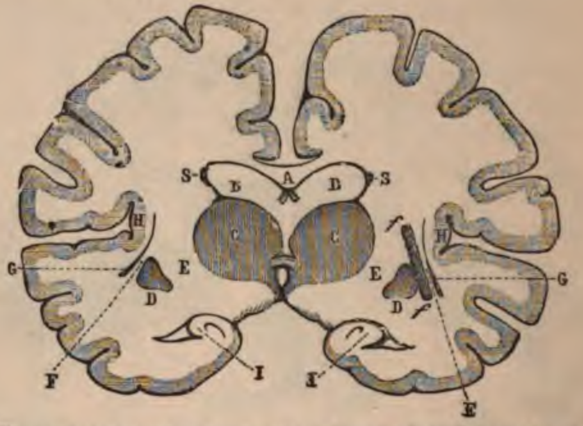

FIG. 64. EXTRA-LENTICULAR HÆMORRHAGIC FOCUS ON A LEVEL WITH THE POSTERIOR PART OF THE THALAMUS—IN THE TERRITORY OF THE OPTO-STRIATE ARTERIES [after Charcot].

(Production of Cerebral Hemianæsthesia).

A, Corpus callosum and posterior pillars of the fornix.
B, B, Cavity of the lateral ventricles. C, C, Thalami.
D, D, Lenticular nuclei. E, E, Posterior or lenticulo-optic region of the internal capsule. F, F, External Capsule. G, G, Claustrum. H, H, Island of Reil. I, I, Cornu Ammonis, and posterior cornu of the Lateral Ventricle. S, S, Posterior extremity of the Caudate Nucleus.
f, *f*, Extra-lenticular hæmorrhagic focus involving the posterior part of the internal capsule.

Where, however, the hæmorrhage, and that one of greater magnitude, occurs in the external capsule or near the internal capsule rather more anteriorly, or where it occurs in the internal capsule itself at the same level, we are likely to have the combination of an incomplete hemiplegia in association with the cerebral hemianæsthesia of a very pronounced type. This may be easily understood, because a large lesion in either of the situations named must somewhat compress the fibres of the pyramidal motor tract (Fig. 65).

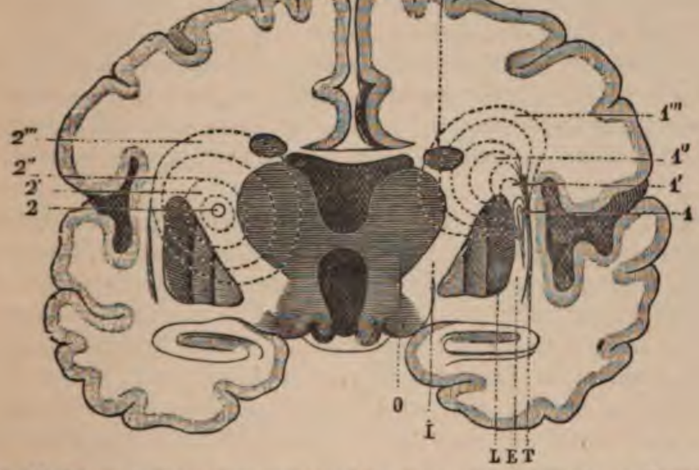

FIG. 65. DIAGRAM ILLUSTRATING SEATS AND MODES OF EXTENSION OF HÆMORRHAGES NEARLY CORRESPONDING WITH THE POSTERIOR THIRD OF THE HINDER SEGMENT OF THE INTERNAL CAPSULE, PRODUCED BY RUPTURE OF DIFFERENT LENTICULO-OPTIC ARTERIES [after Charcot].

(Production of Hemianæsthesia with incomplete Hemiplegia)

O, Thalamus. I, Internal Capsule. L, Lenticular nucleus. E, External Capsule. T, Claustrum. C, Caudate nucleus.

1, Primary focus in the hinder part of the external capsule. $1'$, $1''$, $1'''$, Progressive extension of the primary lesion (compression or destruction of the internal capsule).

2, Primary focal lesion in the posterior third of the hinder segment of the internal capsule.

$2'$, $2''$, $2'''$, Progressive extension of this lesion (destruction of the external capsule, displacement or destruction of the thalamus).

Again, in cases in which the lesion principally compresses or destroys that portion of the internal capsule which gives rise to hemiplegia, a minor amount of pressure may well be exerted upon the hinder or sensory section of the capsule, so as to produce mere numbness or else some slight temporary loss of sensibility, such as occurs so frequently in association with hemiplegias caused by lesions in, or just outside, the internal capsule.

One other important fact in connection with hæmorrhage into the Corpus Striatum or the Thalamus remains to be mentioned, and that is, the liability of hæmorrhagic foci in these situations to break through into the lateral ventricles—an event which, when it happens, is almost sure to favour the further outpouring of blood, and to lead on to a speedily fatal result. There are two arteries in particular the rupture of one or other of which is prone to give rise to this accident, viz., the branch of the anterior cerebral which supplies the caudate nucleus, and the internal posterior optic branch of the posterior cerebral by which the inner aspect of the optic thalamus is supplied. Large hæmorrhages from any one of the lenticulo-striate arteries may, however, at times burst through the caudate nucleus into the lateral ventricles. The signs and symptoms the presence of which may lead us to the diagnosis of such an accident will be referred to further on (p. 316).

Signs and Symptoms of Lesions in the Internal Capsule.—What has been said (*a*) in the last section concerning the blood supply of the internal capsule, and of the pathological accidents to which it is liable; together with (*b*) what has been said at p. 210 in reference to the track of 'secondary degenerations' through its different segments, and (*c*) in the section concerning the production of Cerebral Hemianæsthesia (p. 152), will be found to comprise all the information that is needful in regard to the signs and symptoms attaching to lesions in different parts of the internal capsule. It will not, however, be necessary in practice for the student or practitioner to refer to the second of these sources of information (*b*), since this section merely contains the proof or evidence in support of the localisations assigned in the previous section (*a*).

It should be mentioned, however, that hæmorrhages in this situation, when the blood is poured out suddenly and is not very small in amount, are generally accompanied by loss of consciousness and a more or less distinct apoplectic condition, the duration of which is extremely variable. Speedily, an exaggeration of the deep reflexes, together with early and persistent rigidity, is apt to declare itself in one or both of the paralysed limbs.

Signs and Symptoms of Lesions in the Caudate Nucleus.—Great uncertainty, at present, exists in reference to the signs and symptoms attaching to lesions limited to the caudate nucleus. Almost the same thing has to be said, moreover, in reference to the other two contiguous basal ganglia, viz., the lenticular nucleus, and the thalamus. Symptoms which were formerly believed to be dependent upon damage either to the combined caudate and lenticular nuclei (corpus striatum) or to the thalamus, are now referred with much better reason to lesions in different parts of the internal capsule.

We are left, therefore, with difficult problems to solve concerning the differential signs, if any, that are to be relied upon, for the diagnosis of lesions (such as softenings) in, and confined to, the caudate nucleus, the lenticular nucleus, and the thalamus respectively. The problems are difficult, because of the rarity of softenings precisely limited to one or other of these ganglia, and unassociated with lesions elsewhere. Then, again, in order that such a case (when it occurs, and when it happens to be followed by death as well as by an autopsy) may be of use for furthering our knowledge, it is imperative that it should have been carefully observed during life, and that the brain should have been just as carefully examined after death. Here, as in other cases, where a strict determination of symptomatology is what is aimed at, it is needful to look out for localised softenings, more especially; since a hæmorrhage, sufficiently large to destroy a considerable proportion of either of these ganglia, is apt to cause such an amount of pressure upon contiguous parts (and especially upon the internal capsule itself) as to confuse the clinical picture. The same kind of difficulty presents itself in drawing conclusions from localised abscesses, or from tumours, confined to either of the basal ganglia.

Such considerations fully account for the present uncertain state of our knowledge on these points, and explain the difficulty and delay which is likely to be experienced before our doubts are resolved.

Signs and Symptoms of Lesions in the Lenticular Nucleus.—Seeing that no positive differentiation can be made during life between lesions in the caudate and in the lenticular nucleus, and that the principal diagnostic problem for us, at present, is to distinguish between lesions of the internal capsule and lesions of one or other of these two ganglia, it will be as well to say what can be said upon this subject under the present head, with the understanding that what is said is equally applicable to lesions limited to the caudate nucleus.

Charcot ('Localisation,' p. 80) also declares that it is impossible, during life, to distinguish a lesion limited to the lenticular nucleus from one confined to the caudate nucleus. Lesions of the thalamus, however, now stand a somewhat better chance of being discriminated.

A lesion in either the caudate or the lenticular nucleus is capable of causing an ordinary common form of hemiplegia, without appreciable impairment of sensibility. "Hemiplegia consequent on alterations thus circumscribed in the grey nuclei is, however, generally transitory, ill-defined, non-persistent, and hence comparatively mild" (Charcot). The comparative mildness of lesions limited to the substance of either of the grey nuclei is doubtless referable, in part, to the fact that lesions so limited are commonly small, not damaging the body in question in its entirety. A more severe lesion is, owing to topographical relations and conditions of blood supply, extremely apt also to involve either the internal capsule or one of the other grey nuclei.

Charcot adds:—"On the other hand, the transitory nature of the paralysis resulting from these partial lesions of the central ganglionic masses may indicate, as we shall see, a kind of functional substitution which, in case of necessity, may become established either between the various parts of the caudate nucleus, or between the caudate nucleus and the different segments of the lenticular nucleus."*

The transitory and ill-defined nature of the paralysis resulting from a lesion limited to either the caudate or the lenticular nucleus, seems, then, to be the principal characteristic of such a localisation. The effects of lesions in these situations thus contrast notably with the well-defined and persistent forms of paralysis (apt to be followed by rigidity) which result from lesions of the internal capsule itself.

Signs and Symptoms of Lesions in the Thalamus.—As already stated, nothing very definite can be said concerning the symptomatology of lesions strictly limited to the thalamus. Such lesions are indeed very rare. The thalamus is commonly damaged (*a*) in concert with the internal capsule and parts of the corpus striatum, when (according to the precise situation and extent of the lesion) different sets of symptoms are produced such as have been already indicated (p. 307). At other times (*b*) the thalamus is damaged (by hæmorrhage more especially) in common with the upper part of the crus cerebri (p. 325); here, again, the symptoms are of a mixed order, and are liable to much variation in accordance with the precise seat and extent of the lesion.

Within the last four years I have seen, and had under observation from the day of onset of the attack until death, two cases of hæmorrhage into the right thalamus. In one of them the hæmorrhage was absolutely limited to the thalamus; in the other it involved also the posterior third of the hinder segment of the internal capsule. In this last there was almost complete heminanæsthesia of the opposite side of the body; while in the former case no distinct difference in sensibility could be detected on the two sides of the body.

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* See also p. 213 of this work.

The onset of the attack was marked in each case by vertigo and faintness, with loss of consciousness lasting only a few minutes, but followed by drowsiness and some mental confusion.

In each case, also, there was paresis rather than paralysis of the opposite limbs; the paralysis of the face was very slightly marked; and there was no deviation of the tongue.

Some amount of rigidity of the arm and of the leg was noticed within the first two or three days, and this continued throughout. It was especially noticed in the case in which the hemianæsthesia existed, that the superficial reflexes were not diminished on the affected side, and that the deep reflexes were not exaggerated. The knee-jerk was equal on the two sides and not excessive; whilst of ankle clonus there was not a trace throughout the whole of the two months during

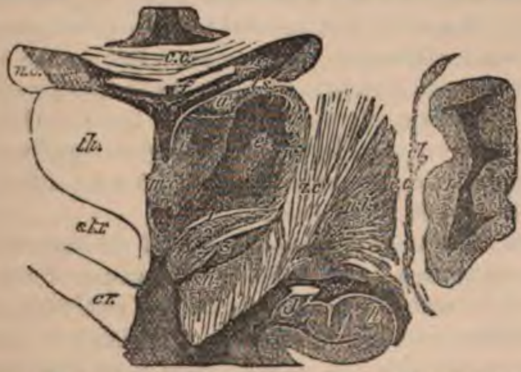

FIG. 66.—SECTION ACROSS THE THALAMUS AND CORPUS STRIATUM IN THE REGION OF THE MIDDLE COMMISSURE, SHOWING SOME OF THE RELATIONS OF THE THALAMUS WITH OTHER PARTS [after Schäfer and Shattock]. Natural size.

th, Thalamus; *a*, *e*, *i*, its anterior, external, and internal nuclei respectively; *w*, its latticed layer.

m.c, Middle commissure, above and below which is the cavity of the third ventricle.

c.c, Corpus callosum; *f*, fornix, separated from the third ventricle and thalamus by the velum interpositum. *t.s*, Tænia semicircularis.

cr, Forward prolongation of the crusta of the cerebral peduncle, passing laterally into the internal capsule (*i.c*).

s.t.r, Subthalmic prolongation of the tegmentum, consisting of (1) the dorsal layer, (2) the zona incerta, and (3) the corpus subthalamicum. *s.n*, Substantia nigra.

n.c, Nucleus caudatus of the corpus striatum; *n.l*, nucleus lenticularis. *e.c*, External capsule. *cl*, Claustrum.

I, Island of Reil.

which the patient lived. The electrical irritability of the muscles was normal.

There was in one case loss of control over the bladder alone, and in the other over both bladder and rectum. [These are the only two cases of unilateral hemiplegia, without very distinct mental impairment, in which I have seen incontinence of urine continue for weeks and months (p. 130); they surprised me much at the time because the amount of mental impairment did not seem sufficient to account for such incontinence].

In both cases there was frequent drowsiness and some amount of mental confusion at times. Sleep was frequently restless, marked by hallucinations (visual and auditory) and much talking. One of these patients not unfrequently awoke in a state of great excitement—swearing, using abusive language, and even attempting to strike the nurse. In from half an hour to an hour these attacks of excitement subsided; no similar attacks occurred during the day time.

I would not lay too much stress upon these two cases—especially as we had in them to do with hæmorrhage rather than with softening. We must wait for others, in order to see whether there will or will not be a tendency to reproduce anything like the same combination of signs and symptoms. It is clear, however, that should this be the case, they will prove an exceedingly characteristic combination whose existence would be capable of leading, without much difficulty, to the diagnosis of a lesion in the thalamus.

The close relations of the posterior part of the thalamus with the optic tract, may be a sufficient explanation of the fact that bilateral hemiopia has occasionally been associated with lesions in this situation. On the other hand, this sign may possibly be a direct consequence of lesions of this portion of the thalamus (Hughlings Jackson).

Hæmorrhage into the Lateral Ventricles.—An accident of this kind is often divided into distinct stages. Thus, we may have at first to do with an ordinary hemiplegic condition—commencing either by an apoplectiform, an epileptiform, or a simple mode of onset. But, after an interval of varying duration, a fresh hæmorrhage occurs, and the blood then poured out may tear its way through the brain substance into the lateral ventricles, so that a condition of profound coma with stertor and general paralysis quickly supervenes. As we have already stated, the hæmorrhage in these cases usually proceeds from the rupture of one of the branches supplied to the caudate nucleus by the anterior cerebral artery, or else from one of the posterior cerebral branches furnished to the thalamus.

In other cases there is no preliminary attack of any kind. A large hæmorrhage occurs suddenly in one or other of the situations named, and the effused blood at once lacerates the brain substance, and opens up a channel through which it is copiously poured into the lateral ventricles. In such a case, we have produced at once an apoplectic attack of the most marked kind, characterized by profound coma, general paralysis of limbs, and dilated pupils.

In each set of cases the laceration of brain tissue and effusion of blood into the ventricles is associated with a decided lowering of the temperature of the body. The rectal temperature sinks to 96°, or thereabouts (perhaps even as low as 94° F.), and may remain at this point for three or four hours—especially where the bleeding into the ventricles slowly continues. Should a fatal result not speedily occur, we find that the primary depression of temperature is followed after a time by a rapid rise, which slowly continues, in cases about to prove fatal, till the death of the patient. Such fatal result may be reached within a few hours; in the great majority of cases it occurs before the expiration of three days from the onset of the attack, but occasionally not till a later period.

In these cases of ventricular hæmorrhage, we very frequently indeed meet with tonic spasms of one, two, or more limbs; or tonic may alternate with clonic spasms in the same parts. In other instances we have a condition of rigidity in the limbs of one side, combined with clonic spasms in one or both extremities of the opposite side. In two recent cases, I met in one, with frequently recurring unilateral convulsions, affecting the paralysed side; while in the other case, in which the blood seems to have been poured out much more rapidly, there was a complete absence of rigidity or convulsions, and rather the existence from the first of a general relaxation of limbs. Concerning the cause of the rigidities or convulsions, when they occur, the views of Pitres have already been referred to (p. 296).

In this class of cases 'conjugate deviation' of the eyes is sometimes present from the first—pointing to the side of the brain on which the hæmorrhage with laceration has occurred. And if the coma is not too profound, the conclusion that may be drawn from this indication may be confirmed by the discovery of some slight signs of sensibility on the side of the body towards which the eyes are turned. Touching the conjunctiva on this side, for instance, may produce some closure of the eyelids, whilst irritation of the conjunctiva on the other side (opposite to that of the brain lesion) gives rise to no corresponding reflex movements.

The grouping of symptoms just described is tolerably distinctive of hæmorrhage into the lateral ventricles. When such symptoms are present we should in the majority of cases be justified in arriving at this diagnosis. More rarely, however, we find such combinations of symptoms pretty closely imitated by the results of lesions in the pons Varolii, when we have to do with injuries, either (*a*) small at first and subsequently increasing, or (*b*) large from the first. In these cases the symptoms may respectively resemble those produced by a slow or by a rapid and copious effusion of blood into one of the lateral ventricles.

In the former class of cases we might be guided to a correct diagnosis if the hemiplegia which had at first existed was of such a nature as to make it referable to a lesion of the pons. And it should be borne in mind that when the condition of coma with general paralysis has become established as a result of an extensive lesion in the pons Varolii (whether this lesion be primary or secondary), the condition itself is very apt to be associated with contracted and motionless pupils, as in opium poisoning, whereas in cases of ventricular hæmorrhage the pupils are usually dilated. (Where large ventricular hæmorrhages occur, however, so that blood passes from the third into the fourth ventricle, and thence beneath the arachnoid, around the medulla and pons, I have seen the pupils notably contracted and insensitive).

Tonic spasms are also more frequently absent in severe central lesions of the pons Varolii than where we have to do with ventricular hæmorrhage. We are apt to find, instead, a complete resolution of all the limbs and of the trunk muscles.

Other cases of brain disease occasionally occur in which the resemblance of the symptoms to those of ventricular hæmorrhage may be extremely close. I allude especially to instances where a hemiplegic condition becomes complicated by the occurrence of a fresh lesion (either softening or hæmorrhage) in the previously sound hemisphere. Here we have the establishment of paralysis on both sides of the body, and generally a condition of profound unconsciousness. I have seen such a case almost exactly simulate one of secondary ventricular hæmorrhage; and, in this instance, the only indication of the real nature of the case (whose importance was not adequately realised at the time), was the simultaneous occurrence of 'conjugate deviation' of the eyes and head towards the previously sound side of the brain.

The power of diagnosing between these conditions is occasionally a matter of some importance from the point of view of prognosis.

PARALYSES DUE TO LESIONS IN THE CORPORA QUADRIGEMINA.

Lesions in these bodies are rare. They are occasionally affected on one or both sides by softening, though hæmorrhage into their substance is extremely uncommon. Tumours occur, not unfrequently, which may originate either in their substance or in that of the overlying pineal body (conarium).

Lesions of the quadrigeminal bodies are marked by varying and often ill-defined symptomatology. This is due in part to the presence of complications, which the existence of disease in these bodies (and especially tumours originating either in them or in the conarium) is apt to involve. Thus, the velum interpositum and the venæ magnæ Galeni may be so much compressed as to lead to effusion into the lateral ventricles, and the consequent production of a new set of symptoms such as pertain to chronic hydrocephalus. Or the pressure of a tumour in this situation, may tell injuriously upon the varied, and important parts which underlie the corpora quadrigemina, such as the upper cerebellar peduncles, the crura cerebri and the pons, or upon one or more of the cranial nerves.

Facts of Importance concerning the Blood Supply of the Quadrigeminal Bodies.—It should be borne in mind that the blood supply, on each side, of the anterior, and of part of the posterior, quadrigeminal body is derived from the posterior cerebral artery; and, moreover, that the same artery supplies the contiguous corpora geniculata and posterior extremity of the thalamus—parts which are endowed with a community of function in regard to vision.

Signs and Symptoms of Lesions in the Corpora Quadrigemina.—The anterior and the posterior quadrigeminal bodies have probably very distinct functions, though our knowledge concerning the exact functions of the posterior pair is at present very defective.

It seems to be a well-established fact that the anterior quadrigeminal bodies contain the centres which co-ordinate retinal impressions with varying sizes of the pupil, through the branch of the third nerve which supplies the iris,* so that destruction of these bodies reduces the pupils to a motionless condition when exposed to light—their size being commonly that of medium or slight dilatation.

Any such destruction would, however, also be associated with visual defects—though as to the precise nature of these defects

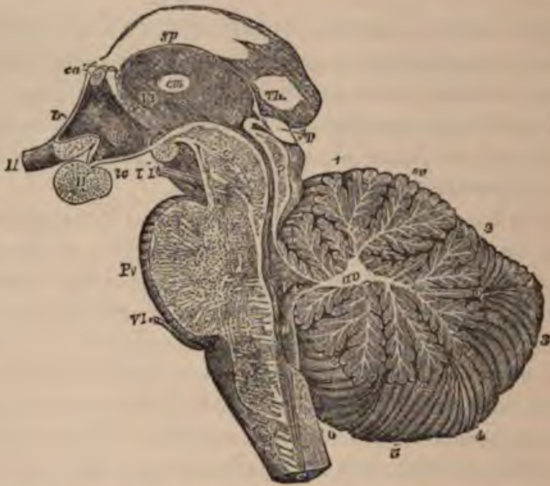

FIG. 67. RIGHT HALF OF THE ENCEPHALIC PEDUNCLE, CORPORA QUADRIGEMINA AND ADJACENT PARTS, AS SEEN FROM A MEDIAN SECTION [Allen Thompson after Reichert].

II, Right optic nerve; behind it the optic commissure divided. III, Right third nerve. VI, Sixth nerve. V3, Third ventricle.

Th, Back part of the Thalamus. H, Section of the Pituitary body. *p*, Pineal body; below its stalk is the posterior commissure. *ca*, Anterior commissure divided, and behind it the divided anterior pillar of the fornix (*f*). *cm*, Commissura mollis. *sp*, Stria pinealis, or peduncle of pineal gland.

lc, Lamina cinerea. *i*, Infundibulum. *tc*, Tuber cinereum; behind it the corpus albicans.

cr, Crus Cerebri. Q, Quadrigeminal bodies. *as*, Aqueduct of Sylvius leading from the third into (V4) the fourth ventricle.

Pv, Pons Varolii. M, Medulla oblongata; and behind these the Cerebellum.

there does not exist any unanimity of opinion, owing to the uncertainty that prevails as to the exact disposition of the optic fibres on their way to the cerebral cortex (see p. 143).

Adopting the view of Charcot, as that which from a survey of all the facts seems to constitute the best hypothesis as to their distribution, we may expect to meet with the following results in the way of visual defect, as a consequence of lesions of the anterior quadrigeminal bodies, either alone or in concert with the corpora geniculata.

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* It should be remembered also that bilateral contraction of pupils may be occasioned by unilateral retinal impressions; so that, in cases of blindness on one side, due to optic atrophy, the corresponding pupil will generally contract (more or less freely) when the sound eye is exposed to light.

(1).—Lesions restricted to the anterior quadrigeminal bodies should produce nasal hemiopia; though by some curious oversight it is stated in Charcot's work ('Localisation,' p. 119) that the result would be the production of crossed amblyopia.

(2).—If one anterior quadrigeminal body together with the external geniculate body of the same side were seriously damaged, or cut off from their blood supply (as may happen for instance in a case of thrombosis of the posterior cerebral artery, which supplies these parts), the result would be the production of a crossed amblyopia plus nasal hemiopia on the side affected. (The same lesion occurring in a person in whom the decussation of the optic fibres had chanced to be total at the optic chiasma, would give rise simply to crossed amblyopia.)

In some lectures published in the 'Lancet' in 1874 and subsequently republished under the title 'Paralysis from Brain Disease,' I stated (p. 113)

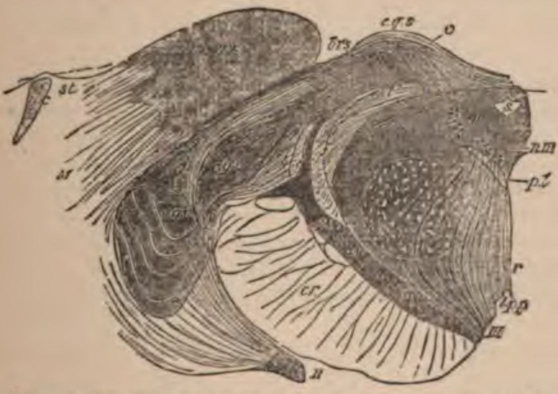

FIG. 68. SECTION THROUGH THE SUPERIOR PART OF ONE OF THE ANTERIOR QUADRIGEMINAL BODIES AND THE ADJACENT PART OF THE THALAMUS [after Meynert].

s, Aqueduct of Sylvius; *gr*, grey matter of the aqueduct. *c.q.s*, Quadrigeminal body, consisting of: *l*, stratum lemnisci; *o*, stratum opticum; *c*, stratum cinereum.
Th, Thalamus (pulvinar).
c.g.i, *c.g.e*, Internal and external geniculate bodies.
b.r.s, *b.r.i*, Superior and inferior brachia.
f, The fillet. *p.l*, Posterior longitudinal bundle. *r*, Raphé.
III, Third nerve; nIII, its nucleus. *l.p.p*, Posterior perforated space.
s.n, Substantia nigra; above this is the tegmentum with its nucleus, the latter being indicated by the circular area. *cr*, Crusta.
II, Optic tract. M, Medullary centre of the hemisphere.
n.c, Nucleus caudatus. *st*, Stria terminalis.

that simultaneous onset of loss, or decided impairment of the sight of one eye with hemiplegia on the same side of the body, afforded a diagnostic indication in favour of thrombosis of the posterior cerebral artery. This observation has been referred to by Charcot and others as unique; whilst Ferrier thinks that such unilateral loss of sight occurring on the hemiplegic side only, and at the same time as the limb paralysis, should rather be attributed to some sudden interference with the cortical visual centre ('Localisation of Cerebral Disease,' p. 132). I am not at present clear what other cases I had in view when the above statement was made. I have, however, found some notes of one such case which came under my observation in 1868 at the National Hospital for the Paralysed and Epileptic.

These notes are deficient in regard to several important points, but seeing that the unilateral blindness was absolute, and had continued so for the three years intervening between the date of the attack and the patient's coming under my observation, it seems perfectly clear that Ferrier's suggestion will not hold good. Broadbent has, moreover, recorded a case in which temporary blindness seems to have been caused by a temporary thrombosis of the posterior cerebral artery of one side ('Clin. Trans.,' 1876, pp. 64 and 67).

(3). Lesions implicating the two anterior quadrigeminal bodies as well as the external geniculate bodies on each side, should produce total blindness. This may happen as a rare event, either from softening or from the growth of a tumour in this situation.

Many years ago I saw a case of complete blindness caused by softening "almost limited to the anterior quadrigeminal bodies." I cannot now say, as a matter of fact, that in this case the softening did actually involve the geniculate bodies as well, though it would seem very probable that this was so. Polyuria was likewise a prominent feature of this case. Lépine quotes a case of tumour of the pineal body in which polyuria was also met with; he says there are other such cases on record, and adds:—"Doubtless, it is not the lesion of the corpora quadrigemina which is to be held responsible, but rather that of subjacent nerves," without saying anything more specific.

As Nothnagel ('Topische Diagnostick der Gehirnkrankheiten,' 1879) points out, the fact that lesions involving the anterior quadrigeminal bodies may produce blindness, is an equivocal fact in itself, and would have no value for leading to the special diagnosis of disease in the anterior quadrigeminal bodies, except in the absence of optic neuritis or optic atrophy. The existence of amaurosis might be considered to have such a special significance (when caused by softening rather than by tumour) if it has supervened in an acute or sub-acute manner, together with dilated and insensitive pupils, and with the absence of morbid changes in the discs as revealed by ophthalmoscopic examination—in other words, such signs might point to the possibility of softening (rather than of tumour) in the anterior quadrigeminal and external geniculate bodies.

In addition to blindness and abolition of the pupillary reflex, Nothnagel speaks of bilateral paresis or paralysis of some branches of the third nerve as being not unfrequently met with in association with disease in the quadrigeminal bodies. This sign, though by no means always present, may, when it exists, be accounted for by the proximity of the nucleus of the third nerve to the lower strata of the quadrigeminal bodies (Fig. 68).

Disturbances in equilibration and locomotion, as well as in emotional expression, are results which have been inferred (from numerous experiments with all classes of animals) as likely to result from lesions of the quadrigeminal bodies, though such results are by no means well established by clinico-pathological investigation.

Ferrier gives a good *résumé* of the experimental evidence ('Functions of the Brain,' pp. 72-84) adduced by others as well as by himself in reference to this question. The subject is an extremely complicated one. He says:—"I make no attempt to differentiate between the optic ganglia, as such, and the underlying tracts, for I do not think it possible to determine experimentally what are the functions of the mesencephalic ganglia and cerebellum, apart from their connections and relations to the crura and pons."

Irritation of these bodies seems to have yielded a very definite and constant series of results in different kinds of animals. Referring to experiments with monkeys, Ferrier says:—"On irritation of the surface of the nates or anterior tubercles in monkeys, I have observed the following results:—Irritation of one side causes the opposite pupil to become widely dilated, followed almost immediately by dilatation of the pupil on the same side. The eyes are widely opened and the eyebrows elevated. The eyeballs are directed upwards and to the opposite side. If the left tubercle is irritated the eyes are directed to the right, and up. The head is moved in the direction of the eyes. The ears are strongly retracted. With continuance of the irritation the tail becomes elevated, the legs extended, and the jaws firmly clenched, with the angles of the mouth retracted to the utmost. The arms are approximated to the sides, and drawn back flexed at the elbows. Ultimately, when the stimulation is kept up, a state of complete opisthotonos is produced. Irritation of the testes or posterior tubercles produces the same effects, but in addition, cries are excited varying in character from a short bark, caused by the slightest contact with the electrodes, to all varieties of vocalisation when the stimulation is continued. The motor effects are shown, first on the opposite side of the body, but ultimately both sides become affected by the unilateral irritation."

Ferrier adds:—"The movements of the trunk and limbs which are produced by excitation of the corpora quadrigemina are such as are concerned in the normal attitude, and for purposes of progression. Irritation of the optic lobes in frogs, excites movements of the head, trunk and limbs; in fishes, causes movements of the tail and fins; in birds, causes movements of the wings; and in mammals, complex movements of the trunk and extremities."

PARALYSES DUE TO LESIONS OF THE CRUS CEREBRI.

The crus cerebri is a well defined segment of the brain whose situation and boundaries need not be particularly described; it will only be necessary to remind the student that the anterior quadrigeminal bodies rest upon the upper or hinder part of the crura; that just beneath these bodies and the Sylvian aqueduct the nuclei of the third and fourth nerves are situated; and that from these nuclei the root fibres

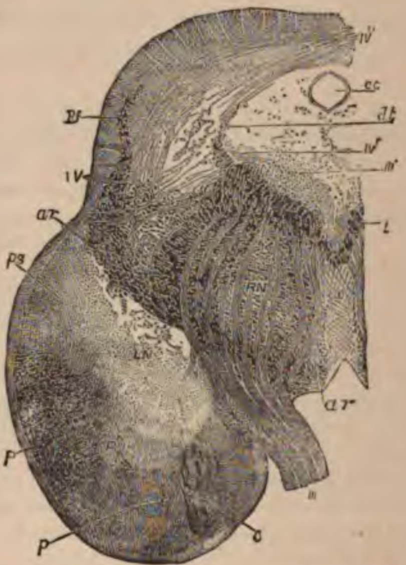

FIG. 69. TRANSVERSE SECTION OF THE CRURA CEREBRI ON A LEVEL WITH THE ANTERIOR PAIR OF CORPORA QUADRIGEMINA AND THE ROOTS OF THE THIRD NERVE, FROM A NINE-MONTHS EMBRYO [after Krause and Ross].

C, C, Crusta. P, Pyramidal tract; *p*, accessory portion of the pyramidal tract.
LN, Locus niger. RN, Red nucleus of the tegmentum. L, Posterior longitudinal fasciculus. *ar* and *ar'*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord.
III, Third nerve; III', nucleus of the third nerve.
IV, Fourth nerve; IV', nucleus of the fourth nerve. IV'', Crossing of the fibres of the fourth nerves to opposite sides.
dt, Descending root of the trigeminus. *cc*, Aqueduct of Sylvius. *x*, Crossing of the fibres of the superior peduncles of the Cerebellum. *pf*, Fasciculus of medullated fibres proceeding to the anterior pair of Corpora Quadrigemina.

of the third nerves are directed downwards and forwards making their appearance at the base of the brain on the inner side of each cerebral peduncle.

Facts of importance concerning the blood supply of the Crus Cerebri.—The crus cerebri is wholly supplied with blood by the posterior cerebral artery. A thrombosis affecting the first part of this artery will, therefore, cut off the blood supply from the corresponding peduncle, and so produce, as part of a larger set of symptoms, those also which are about to be described as consequences of a lesion in the crus.

Rupture or occlusion of one or more of the small twigs of the basal system given off from the posterior cerebral to the crus, will cause symptoms more limited in range, and referable to larger and smaller lesions in this brain region.

The larger branch of the posterior cerebral, known as the posterior external optic artery, which is given off after the main vessel has bent round the crus cerebri often ascends through its substance so as to penetrate the posterior part of the thalamus. When this artery is ruptured, it often gives rise to a large hæmorrhage into this region of the thalamus which extends thence into the substance of the cerebral peduncle, perhaps throughout its whole length. In any of these cases, if the patient should recover from the apoplectic condition, there would almost certainly be a hemiplegia associated with marked hemianæsthesia on the paralysed side, but probably without any distinct paralysis of the opposite third nerve.

Signs and Symptoms of Lesions in the Crus Cerebri.—The crus cerebri may, in regard to diagnosis, be divided into two parts along an imaginary diagonal line, viz., into (*a*) a lower and inner portion, and (*b*) an upper and outer portion. A lesion in the former situation can generally be diagnosed with much certainty; though the same cannot be said in regard to lesions in the latter brain region.

a.—Lesions in the Lower and Inner Portion of the Crus.—A lesion in this part of the crus causes the simultaneous occurrence of paralysis of the third nerve on the side of the lesion together with a hemiplegic condition of the opposite half of the body. We have here the production of a typical form of 'alternate paralysis,' of great diagnostic significance.

More in detail the signs are these. On one side ptosis, pupil of medium size and insensitive to light, external squint, and an impossibility of moving the eyeball except slightly in two directions—viz., slightly further outwards (owing to forced contraction of the external rectus), and a little around its own axis in one direction; that is, from outwards upwards (owing to forced contraction of the superior oblique muscle). All the muscles of the eyeball, in short, are paralysed, except the external rectus and the superior oblique, which are supplied by the sixth and fourth nerves respectively.

The coexisting hemiplegia on the opposite side of the body approximates in its general characters to that produced by a lesion in the upper part of one lateral half of the pons. The tongue generally deviates distinctly to the paralysed side, and the facial paralysis about the mouth is well marked. Aphemic defect may be well marked, or slight; and deglutition may not be much interfered with. Sensibility may or may not be decidedly impaired on the paralysed side, according as the lesion does or does not involve the outer part of this peduncular region. The temperature of the non-paralysed side may be as much as two degrees lower than that of the paralysed side of the body.

b.—Lesions in the Upper and Outer Portion of the Crus.—There is no distinctive sign of a lesion in this situation. The grouping of symptoms would approximate very closely to that met with as a result of lesions of the posterior part of the internal capsule, just outside the posterior extremity of the thalamus, and could not well be distinguished from them. In each case there would be the combination of a well-marked hemianæsthesia together with a slightly marked motor paralysis, of the opposite side of the body. A lesion of this part of the crus has no special tendency to produce paralysis of the third nerve on the side of lesion, and, consequently, it is not marked by any distinctive characters.

III.—LESIONS IN PARTS SUPPLIED BY THE VERTEBRAL AND BASILAR ARTERIES.

DISTRIBUTION OF THE VERTEBRAL AND BASILAR ARTERIES.

It remains for us now to say a few words concerning the vascular supply of the **Cerebellum**, the **Pons**, and the **Medulla Oblongata**—parts of the encephalon which are all supplied from the vertebrals and their prolongation, the basilar artery.

Arterial Supply of the Cerebellum.—The little brain, like the cerebrum is supplied by three vessels on each side. These vessels, however, unlike those of the cerebrum, are said to freely anastomose on the surface of the cerebellum. From this network multitudes of minute twigs penetrate into the substance of the organ.

The inferior cerebellar arteries arise from near the end of the vertebral, or sometimes from the commencement of the basilar artery; they wind round the upper part of the medulla oblongata (where they give off the posterior spinal arteries) and reach the under surface of the cerebellum, to which they are distributed, as well as the posterior part of its upper surface, where they anastomose with terminal twigs of the superior cerebellar.

The middle cerebellar arteries are given off from the middle of the basilar artery. They run parallel with the transverse arteries of the pons, pass over the middle peduncles, and are distributed over the anterior part of the under surface of the cerebellum.

The superior cerebellar arteries are derived from the basilar close to its termination; they wind round the crus cerebri on each side, and are distributed over the upper surface of the cerebellum.

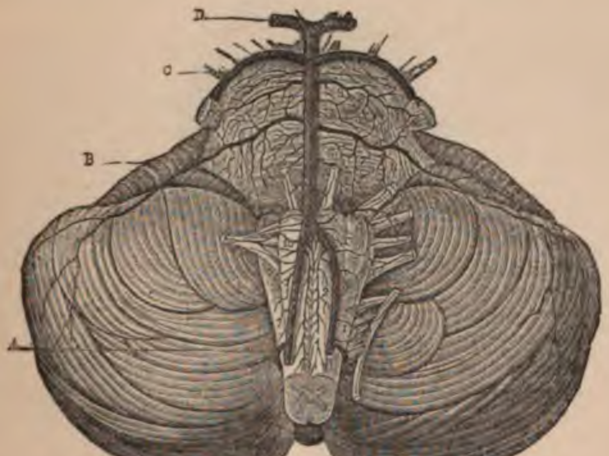

FIG. 70. ARTERIES OF THE MEDULLA OBLONGATA, PONS, AND INFERIOR SURFACE OF THE CEREBELLUM [after Duret].

A, Inferior cerebellar artery. B, Middle cerebellar artery. C, Superior cerebellar artery. D, Posterior cerebral artery.

1, Root arteries of the spinal accessory nerve. 2, Anterior spinal arteries. 3, Root arteries of the pneumo-gastric and glosso-pharyngeal nerves. 4, Inferior root arteries of the auditory and facial nerves (vertebral branches). 5, Root arteries of the sixth nerve. 6 and 7, Arteries of the subolivary fossa. 8, Superior root arteries of the auditory and facial nerves (branches of the middle cerebellar artery). 9, Root arteries of the trigeminal nerve. 10, Root arteries of the hypoglossal nerve (branches of the vertebral and anterior spinal arteries).

Very large hæmorrhages are occasionally met with in the cerebellum, and these, as Duret points out, are due to the rupture of a large artery derived from the superior cerebellar, which enters the grey nucleus (corpus rhomboidale) on each side by its hilum, and there breaks up into radiating branches for the supply of this organ and of the white substance around. Ruptures of this vessel may produce so large an extravasation of blood that it occupies the whole of the corresponding lobe or, more rarely still, nearly the whole of the cerebellum. A rupture of one of its radiating branches for the most part gives rise to a small extravasation contained within the rhomboidal nucleus itself. Hæmorrhages into the white substance outside this body rarely exceed an almond in size.

The superior and inferior peduncles of the cerebellum only contain vessels a little larger than capillaries, and are very rarely the seats of hæmorrhage. The middle peduncle is, however, supplied with larger vessels, derived almost exclusively from the middle cerebellar artery, and here larger extravasations of blood are also occasionally met with, which, following a longitudinal direction, separate the fibres of the middle peduncle and may reach as far as the pons.

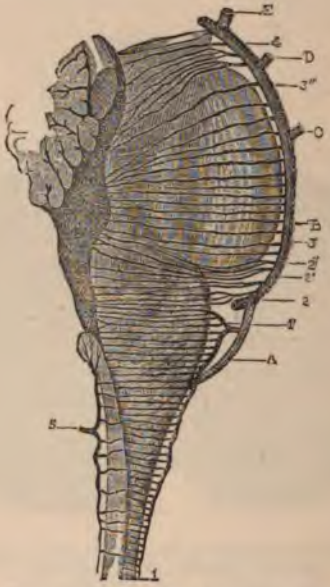

FIG. 71. MEDIAN LONGITUDINAL SECTION THROUGH THE PONS AND MEDULLA, SHOWING THE DISTRIBUTION OF SOME OF THEIR PRINCIPAL ARTERIES [after Duret].

A, Left veretebral artery. B, Basilar artery. C, Middle cerebellar artery. D, Superior cerebellar artery. E, Posterior cerebral artery.
1, 1', Anterior spinal artery, the bulbar branches. 2, 2', 2'', Inferior arteries of the pons. 3, 3', Median arteries of the pons. 4, Superior arteries of the pons. 5, Posterior spinal arteries, median branches.

Arterial Supply of the Pons Varolii and Medulla Oblongata.—The pons Varolii is very richly supplied with minute vessels which penetrate into its substance from the basilar artery—some directly along the middle line, and others, after a short course on either side, into its lateral regions. All these vessels pursue more or less parallel paths towards the upper strata of the pons and the upper half of the floor of the fourth ventricle. They are all typical 'end' arteries. (Figs. 70 and 71 convey an accurate notion as to the distribution of these vessels).

The basilar also supplies certain root arteries, which proceed to the roots of the third, fifth, sixth and seventh nerves.

The arterial supply of the medulla oblongata has been thoroughly investigated by Duret. All its nourishing twigs are derived from the vertebrals. They are divisible into three classes:—(1) The root arteries are lateral, and distributed principally to the nerve roots. (2) The median arteries resemble the similar vessels met with in the pons; they penetrate in the median planes of the medulla to the floor of the fourth ventricle and are distributed to the nerve nuclei and nerve roots in this situation. (3) The vessels of the third class are distributed to the other constituent parts of the bulb, such as the olivary bodies, the pyramids, the restiform bodies, etc.

(1). The root arteries are minute twigs from $\frac{1}{75}''$-$\frac{1}{100}''$ in diameter which arise either from the basilar, the vertebrals, or occasionally from one of the cerebellar arteries; they go to the nerve roots and penetrate them near their points of emergence from the pons or medulla. Here they constantly divide into two branches - one *ascending* which makes for the nuclear origin of the root, and one *descending* which passes along the trunk of the nerve. The ascending branches divide into three or four twigs that pursue a parallel course and at the nucleus of origin resolve themselves into a fine capillary network which inosculates with that of the median twig supplied to the same nerve root.

The root arteries of the third nerves are derived from the upper extremity of the basilar. The derivation of those supplying the nerves which arise from the pons and medulla are stated in the explanation of Fig. 70. The nourishing arteries of the first, second and fourth nerves are given off from branches of the circle of Willis.

(2). The median arteries are especially destined for the nerve nuclei which occupy the floor of the fourth ventricle. Duret divides them into four groups (shown in Fig. 71), viz., (*a*), the proper bulbar arteries derived from the anterior spinal vessels; (*b*), the inferior arteries of the pons (sub-protuberantial of Duret), which are given off from the lower bifurcation of the basilar; (*c*), the median arteries of the pons, derived from the trunk of the basilar; (*d*), superior arteries of the pons, derived from the upper bifurcation of the basilar artery.

All these vessels spring as small twigs (from $\frac{1}{100}''$-$\frac{1}{150}''$ in diameter) direct from a large trunk; they all follow a parallel course in the median plane of

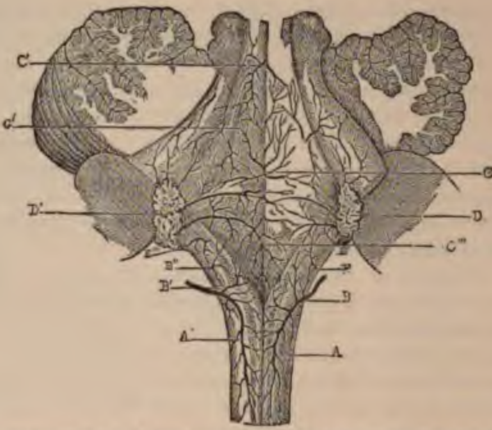

FIG. 72. DISTRIBUTION OF ARTERIES ON THE FLOOR OF THE FOURTH VENTRICLE [after Duret].

A, A', Posterior spinal artery (branch of the inferior cerebellar). B, B', its pyramidal branches.
C, C' C'' C''', Median arteries after their emergence.
D, D', Choroid plexus drawn aside, with arteries emerging from it.
E, E', E'', E''', Arteries of the restiform bodies derived from the inferior cerebellar artery.

the bulb to the floor of the fourth ventricle; they do not anastomose with one another and are true 'end' arteries.

The proper bulbar arteries (*a*) penetrate the medulla, and, like the inferior arteries of the pons (*b*) proceed towards the floor of the fourth ventricle, over the lower half of which they are distributed. Besides these median twigs, the anterior spinal artery gives off minute filaments, which unite with other branches derived from the vertebral, to supply important parts in the lateral regions of the medulla, viz., the hypoglossal roots, and the inferior parts of the pyramids and olivary bodies.

The median arteries of the pons (*c*) are generally slightly larger than their fellows; they converge slightly as they proceed towards the floor of the fourth ventricle (Fig. 71).

The superior arteries of the pons (*d*) pass at its upper border between the cerebral peduncles and the third nerves obliquely backwards and upwards towards the upper angle of the fourth ventricle.

The probable relations of these arteries to the **nerve nuclei** is said by Duret to be as follows:—

1. The branches of the anterior spinal correspond with the inferior regions of the bulb; they supply the spinal accessory, the hypoglossal, and the inferior facial nucleus of Clarke.

2. The inferior arteries of the pons (sub-protuberantial of Duret) correspond especially with the important nuclei of the pneumo-gastric, of the glosso-pharyngeal, and of the auditory nerves.

3. The other median arteries are distributed especially to the upper facial nucleus, and to those of the sixth, third, and fourth nerves.

The disposition within the pons and medulla of all these median arteries is practically the same. Each terminates in two or three twigs which ramify beneath the ependyma of the fourth ventricle. In the first part of their course, these arteries give off a sparing number of branches, which unite with collateral twigs to form an open capillary net work. As they approach the nuclei in the floor of the fourth ventricle they give off two or three larger branches on each side, which make for the nerve nuclei, and there break up into a close capillary network which unites with that formed by the terminal twigs of the ascending branch of the corresponding root artery. The nerve cells of the nuclei are contained within the close meshes of this capillary network.

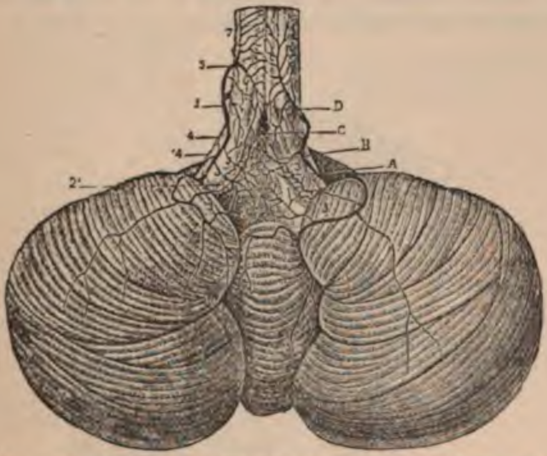

FIG. 73. ARTERIES OF THE POSTERIOR PART OF THE MEDULLA AND OF THE CEREBELLUM [after Duret].

A, Choroid plexus. B, Choroid velum. C, Posterior opening (foramen Majendie) forming a communication between the fourth ventricle and the posterior subarachnoid space. D, Posterior pyramid.

1, Inferior cerebellar artery. 2, 2', Artery of the choroid plexus.

3, 3, 3, 3, Arteries of the choroid velum. Some proceed to the floor of the fourth ventricle; they are capillary.

4, 4', Arteries of the restiform body.

5, Posterior spinal artery; 6, its ascending or pyramidal branch; 7, its descending branch; 8, its median branch.

From the above description it will be seen that each nerve nucleus has two sources of blood supply. Of these, that from the median arteries is by far the most important.

(3). The lateral arteries, or those which supply the other constituent parts of the bulb (the olivary bodies, pyramids, restiform bodies, etc.), constitute the third class of bulbar arteries.

The arteries of the anterior aspect of the medulla, viz., for the pyramids and olivary bodies, come either directly from the vertebrals, through the intermediation of the root arteries which may supply collateral branches, or else in part from twigs of the anterior spinal arteries. Some of the filaments for the olivary body follow the roots of the hypoglossal and enter the olive by its hilum where they are distributed in a radiating fashion.

The arteries of the lateral regions of the medulla are derived from the inferior cerebellar as it turns round to the posterior aspect of the medulla (Fig. 73). It here gives off ascending and descending branches which supply the lateral and intermediate columns and the restiform body.

The arteries of the posterior aspect all come from the inferior cerebellar. The principal of these branches is the posterior spinal artery. This and other filaments are shown in Fig. 71.

Further details concerning the distribution of the three sets of arteries within the medulla may be gathered from an examination of Fig. 74.

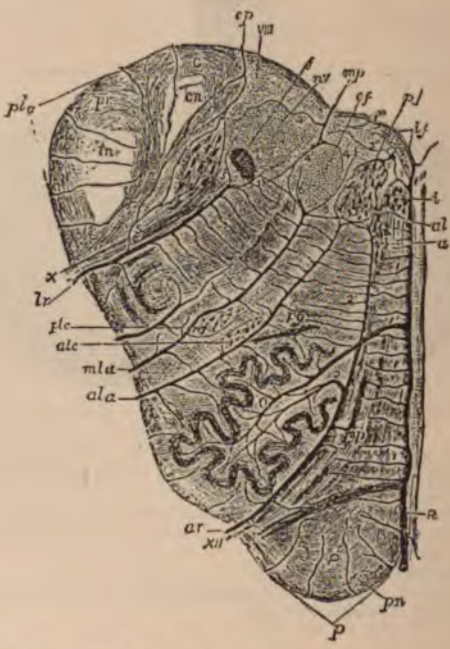

FIG. 74. SECTION THROUGH ONE HALF OF THE MEDULLA OBLONGATA SHOWING THE DISTRIBUTION OF ITS ARTERIES [after Young and Ross].

R, Artery of the median raphé.
I, I, I, branches to the formatio-reticularis.
1′, branch to the olivary body. 1″, branches to the hypoglossal nucleus.
1‴, branches to the floor of the fourth ventricle, and to the internal inferior nuclei of the facial (*if*).

p, pyramidal arteries. *ar*, anterior root artery (hypoglossal).
2′, branch to the olivary body.
2″, branches to the formatio reticularis. (The vessel may be seen to terminate in branches to the hypoglossal nucleus.)
lr, lateral root artery (of the pneumo-gastric).
5, branch to the restiform body and the inner division of the inferior cerebellar peduncle.
5′, branches to the nucleus of the vagus. It also gives branches to the ascending root of the fifth and the formatio reticularis.
ala, anterior lateral artery of the medulla oblongata. It supplies branches to the formatio reticularis, olivary body, anterior nucleus of the lateral column (*alc*), and terminates in branches to the hypoglossal nucleus.
mla, middle lateral artery of the medulla oblongata. It supplies branches to the formatio reticularis, the posterior nucleus of the lateral column (*plc*), and terminates in branches which are distributed to the external accessory nucleus of the facial (*ef*).
pla, posterior lateral arteries of the medulla oblongata. They supply the restiform bodies.
C, Central artery.
3, 3′, 3″, branches to the hypoglossal and external accessory facial nuclei.
mp, median posterior artery.
4, 4′, 4″, branches to the external accessory facial, and pneumo-gastric nuclei.
ep, external posterior artery. It supplies branches to the internal division of the inferior peduncle of the cerebellum and restiform body.
i, internal group of cells of the hypoglossal nucleus.
al, antero-lateral " " "
pl, postero-lateral " " "
a, anterior " " "
VIII, inferior portion of the posterior median auditory nucleus.
if, internal accessory facial nuclei.
ef, external accessory facial nucleus.
f, fasciculus rotundus.
XII, hypoglossal nerve.
X, pneumo-gastric nerve.
G, column of Goll.
pr, posterior root zone. The direct cerebellar tract forms a thin band lying external to the column of Goll and the posterior root zone.
cn, clavate nucleus. *tn*, triangular nucleus. *o*, olivary body. *po*, parolivary body.
np, nucleus of the pyramid. *pn*, nucleus of the arciform fibres.
P, anterior pyramid.

PARALYSES DUE TO LESIONS OF THE PONS.

We include under this term the whole thickness of the brain segment lying between the cerebral peduncles above and the bulb below. On the upper and posterior aspect of this brain region we have the upper half of the fourth ventricle (anterior to the level of the striæ medullares), together with the floor of the Sylvian aqueduct; structures in which are contained several nerve nuclei. In the latter situation, we have the nuclei of the third and fourth nerves; and in the former, part of those of the fifth as well as the sixth and seventh nerves (see Fig. 79).

Facts of importance concerning the blood supply of the Pons.—The median branches are the most important of the secondary arteries of the pons. Rupture of one or other of them gives rise to the central hæmorrhages which are not uncommon in this brain segment. At other times, however, hæmorrhage into the pons occurs from one of the small arteries that penetrates the substance of this body on either side, when different symptoms are produced according as the bleeding takes place into the upper or into the lower half of one lateral region.

Small foci of softening may also be produced by thrombosis of some one or more of the small vessels in the central or lateral regions of the pons. Such foci would have a wedge-like shape, with the base in the direction of, and not far beneath, the floor of the fourth ventricle.

The effects produced by thrombosis of one of these small vessels are likely to be far less marked than those occasioned by its rupture; in the former case a portion of brain substance, rather less than that nourished by the particular artery, would have its functions annulled; while, in the case of the rupture of one of these vessels, the amount of blood extravasated may be sufficient to lacerate and very injuriously press upon portions of the pons altogether outside the territory of the ruptured artery. This difference between the effects of hæmorrhage and thrombosis, is nowhere of more significance than it is in the pons, owing to the functional importance of this segment of the brain, and the great concentration of centres and of sensory and motor channels therein. It may happen here, as well as elsewhere, that hæmorrhage from a secondary or even a tertiary vessel occasionally produces effects as grave as those which would follow from embolism or thrombosis of the parent trunk.

As Duret pointed out, thrombosis of the basilar artery may produce two sets of symptoms differing from one another in a rather marked manner according to the exact situation of the occlusion, that is, according as the portion of the vessel next the bulb, or (this being free) the half of the vessel next the cerebral peduncles, is the portion that has become occluded.

Occlusion of the half of the vessel next the bulb is spoken of under that section (p. 368) because the most important secondary vessels to become occluded in this case are the sub-protuberantial arteries given off from the very commencement of the basilar opposite the upper confines of the bulb (Fig. 71).

Occlusion of the upper half of the basilar gives rise to far less urgent symptoms and is much less likely to prove rapidly fatal. The respiratory centres are not then specially implicated; on the contrary that portion of the basilar is blocked from which the small arteries are given off that make their way to and supply the nuclei of the third, of the sixth, and of the main part of the facial nerves. We may have, therefore, in such a case strabismus of varying character, more or less complete ptosis, and paralysis of the upper facial muscles especially, in association with other signs (notably some amount of bilateral paralysis of limbs) characteristic of a lesion in the pons Varolii.

In the rare cases in which embolism of the basilar artery occurs, it is most prone to be associated with symptoms of this latter type, because an embolus which passes through the vertebral artery is most likely to lodge at the upper extremity of the basilar. It probably will not at first completely occlude the basilar artery, though, after a time, complete occlusion may be brought about by the formation of a thrombus upon the embolus. In such a case there would be a progressive increase in the gravity of the symptoms, after those due to the first abrupt onset of the illness.

An aneurysm of the basilar artery will, of course, press upon and irritate the pons to some extent, but this may give rise only to somewhat vague and indefinite symptoms. Should rûpture occur we may expect rapid death with all the signs of a large meningeal hæmorrhage. In its earlier stages the presence of an aneurysmal dilatation may favour the occurrence of thrombosis, as in a case which I have elsewhere recorded (' Clin. Trans.,' 1885).

Signs and Symptoms of Lesions in the Pons.—Beyond the differences that have been hinted at above in connection with thrombosis of the upper and lower parts of the basilar artery respectively—that is, the involvement especially of the upper and lower median portions of the pons—we have now to mention the principal effects of lesions (*a*) in its central region; (*b*) in its lower lateral region; and (*c*) in its upper lateral region.

(a). *Lesions in the Central Regions of the Pons.*—Large lesions here give rise to profound 'apoplectic' symptoms—characterized by deep coma, complete resolution of limbs on both sides, flapping of cheeks during expiration, insensibility of conjunctivæ (loss of conjunctival reflexes), and more or less markedly contracted pupils. With hæmorrhages in this situation (especially when a moderately large quantity of blood is poured out suddenly) death may take place in a few minutes, though it is more likely not to occur till some few hours have elapsed. With a smaller bleeding, and when the blood is poured out more slowly, the patient may live for two or three days. When there is a fatal issue in the course of a few hours, the patient remains in a state of collapse, and probably with a rectal temperature varying between 96° and 94° F. But where the life of the patient is prolonged from twelve hours to three or four days before the affection proves fatal (and that whether we have to do with hæmorrhage or thrombosis of the basilar artery), after a period of initial collapse with depression of temperature there may be a period in which the body-heat slowly rises, so that at the time of death the rectal temperature may stand at some point between 104° and even 110° F.

During all this time a condition of the profoundest coma, with flaccidity of all muscles and complete abolition of all reflexes, is generally met with. The condition of the pulse will probably vary much from time to time; towards the close it is apt to become extremely irregular and rapid. The respirations too are liable to vary much; they may for a period be slow and irregular in rhythm (possibly of the Cheyne-Stokes type); while at other times, or in other cases, they may be extremely rapid (even as frequent as 105 per minute).

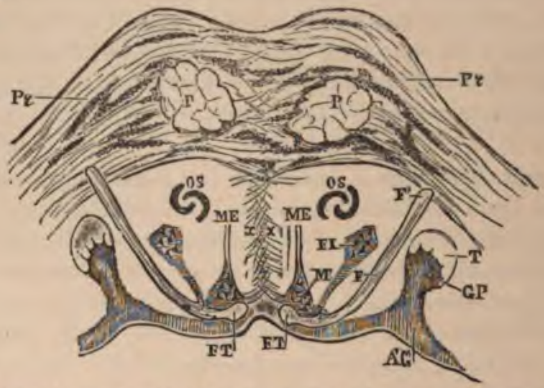

FIG. 75. DIAGRAM OF A TRANSVERSE SECTION THROUGH THE LOWER BORDER OF THE PONS, SHOWING THE RELATIVE POSITIONS OF THE PYRAMIDAL TRACTS AND THE ROOTS OF THE SIXTH AND SEVENTH NERVES [after M. Duval].

P,P, Pyramids. Pr, Pr, Transverse fibres of the pons with interspersed masses of grey substance. CP, Head of the posterior cornu. T, Ascending root of the trigeminal nerve. ME, ME, Roots of the sixth nerve. M, Nucleus of the sixth nerve.

FT, FT, Upper part of the fasciculus teres, or root of the facial which winds round the nucleus of the sixth, and is then directed forwards (F,F') towards its point of emergence, receiving also some root fibres from the inferior nucleus FI.

OS, Superior Olivary Body. A'C', Nucleus of the Auditory Nerve.

When a central lesion in the pons is slighter in extent, the patient after a time recovers from the first shock of the injury, and consciousness is gradually regained. In such cases, we may then find a generalised paralysis more or less equally distributed over the two sides of the body; sensibility also may be very notably diminished or perverted in one or more of the limbs (though it is not likely to be so on both sides of the body). When, in addition to such signs, there is well-marked but irregular paralysis about the face, involving the orbicularis palpebrarum as well as the mouth; when the tongue is notably paralysed, and there is difficulty in deglutition, associated with a decided impediment in articulation (to the extent, it may be, of making the utterance quite unintelligible), we may feel reasonably sure that we have to do with a lesion involving the central parts of the pons Varolii.

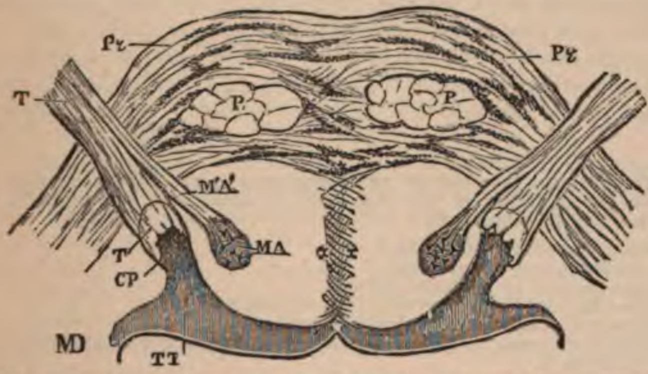

FIG. 76. DIAGRAM OF A SECTION THROUGH THE PONS AT THE LEVEL OF THE EMERGENCE OF THE FIFTH NERVES [after M. Duval].

P,P, Pyramidal strands. Pr, Transverse fibres of the pons with interstratified masses of grey matter. TT, Grey substance of the floor of the fourth ventricle (locus cæruleus). CP, Gelatinous substance of Rolando. T, Ascending root of the trigeminus, curving so as to emerge as part of the sensory root of this nerve. MA, Motor nucleus of the trigeminus. M'A', Motor root of the trigeminus (masticatory nerve). T', The Fifth Nerve at its point of emergence from the pons.

Of course many sub-varieties of this particular type of paralysis are to be met with, though in each of them we may recognize the characteristic combination of irregular bilateral motor paralysis of the limbs and face, well-marked diminution or alteration of sensibility in some parts of the body, together with some distinct difficulty in deglutition as well as in articulation.

(b). *Lower Half of one Lateral Region of the Pons.*—An injury of this part is characterized by the production of what is called 'alternate hemiplegia' or 'cross paralysis' in which we have an unusually well-marked facial paralysis on the side of the brain lesion (owing to the implication of the roots of this nerve), and a more or less complete motor and sensory paralysis of the limbs on the opposite side of the body. Such a hemiplegia may commence with apoplectic symptoms, or there may be an epileptiform mode of onset; whilst in other cases it supervenes more gradually, without either loss of consciousness or convulsions. After the effects of the first shock have disappeared, the temperature of the limbs on the side of lesion is generally found to be about 2° F. lower than it is on the paralysed side. The difficulty in articulation will probably not be very marked; and deglutition may not be much interfered with. The facial paralysis on the side of lesion is notable for its completeness, and because the paralysed muscles may show notably altered electrical reactions soon after its onset. A more or less perfect 'reaction of degeneration' is apt to reveal itself within a short time.

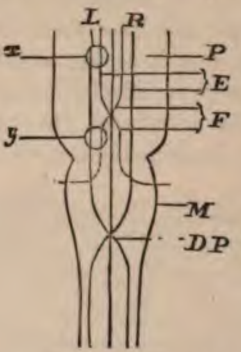

Fig. 77. Diagram Illustrating the Decussation of the Cerebral Fibres for the Facial Nerve in the Pons, and of Fibres for the Limbs in the Medulla [after Nothnagel].

P, pons. M, medulla oblongata.
DP, decussation of the pyramids.
F, fibres destined for the facial nerves.
E, fibres destined for the nerves going to extremities.
x, lesion in the upper lateral half of the pons.
y, lesion in the lower lateral half of the pons (which would be productive of a 'cross paralysis').

(c). *Upper Half of one Lateral Region of the Pons.*—Lesions in this part of the pons give rise to a hemiplegia of the same kind as that last described, with the sole exception that the well-marked paralysis of the face exists on the side opposite to the brain lesion—that is on the same side of the body as the paralysis of the limbs ; for here the fibres of the facial are implicated above their point of decussation in the pons, just as the motor channels for the limbs are implicated above their decussation in the bulb. The fifth nerve may, however, be implicated on the same side as the lesion—especially its motor root (Fig. 76, M'A'). This is a sign that should be looked for, and in certain cases its recognition may serve to confirm an otherwise doubtful diagnosis of the existence of a lesion in the corresponding upper lateral region of the pons.

Whether the lesion be in the upper or in the lower half of one lateral region of the pons, the facial paralysis is generally very well marked—involving the orbicularis palpebrarum as well as the orbicularis oris and the straight muscles going to the angle of the mouth. It is frequently associated with some distinct difficulties in deglutition and articulation, and there is often a very copious overflow of saliva from the paralysed side of the mouth—partly owing to an increased secretion, and partly to the fact of the paralysis favouring its escape and impeding its deglutition.

The degree of impairment of sensibility on the paralysed side of the body is very variable, this symptom being more marked according as the lesion approaches near to, or actually involves, one side of the ventricular aspect of the pons, when we may have the production of a hemianæsthesia of the mæsocephalic tpye (p. 153). Where such an affection of sensibility exists, it is apt to be more durable and more marked than in the great majority of cases of hemiplegia due to lesions further away from the base of the brain. This statement does not hold good, however, for any lesion higher up which chances to involve the posterior third of the hinder segment of the internal capsule (p. 152). More rarely a condition of unilateral hyperæsthesia (often limited in its distribution) may occur instead of anæsthesia ; and either state may be associated with painful sensations in the limbs or with peculiar subjective sensations of 'coldness,' even when the temperature of the part is actually higher than natural.

The fifth nerve is frequently implicated in these cases of lesion in the lateral region of the pons, so that we may have anæsthesia, hyperæsthesia, painful, or anomalous sensations on the corresponding side of the face. There will, moreover, be a weakening of the temporal, masseter, and other muscles of mastication on the same side, if the motor root or nucleus of the fifth be damaged or in any way interfered with.

In addition to the presence of the before-mentioned symptoms in different modes of grouping, lesions of the pons are especially apt to be associated with what is commonly known as 'emotional weakness.' There is an undue proneness to burst into tears or laughter without adequate cause. The tendency to cry is generally more marked than the tendency to laugh. The frequency of this sign is not so much to be wondered at, when we consider that the pons is a centre under whose influence the external manifestations of emotional states are regulated.

Where lesions of the pons cause irritation of parts of the surface of the fourth ventricle we may find sugar in the urine. In other cases there may be polyuria (diabetes insipidus), or albuminuria, when lower portions of the fourth ventricle are implicated—though such symptoms are due to encroachment of the lesion, or of some effects of the lesion, upon the contiguous region of the bulb, and not, apparently, to lesions strictly limited to the pons itself.

While suddenly occurring and extensive lesions in any part of the pons always produce an apoplectic attack of a profound character, slighter lesions are not unfrequently ushered in by an epileptiform onset; though in other instances (and especially where the damage occurs slowly) there is neither loss of consciousness nor convulsions.

Early rigidity or spasmodic states of the paralysed limbs are also very frequently encountered where we have to do with lesions in the pons. But in addition to a spasmodic condition of the muscles of one or both limbs on the paralysed side, there may be trismus, or rigidity of some of the muscles of the neck.

When conjugate deviation of the eyes and head occurs with the onset of apoplectic symptoms due to an acute lesion in the pons, the deviation is towards the paralysed limbs (see p. 64), rather than towards the side of lesion—which is the habitual direction of the deviation caused by paralysing lesions existing in the cerebral hemispheres themselves (when this sign happens to be present).

According to Nothnagel there is one region of the pons the irritation of which is especially prone to give rise to convulsions (and which has consequently been somewhat absurdly spoken of as a 'convulsion centre'). The limits of this irritable region have been pretty accurately defined, by means of experiments, in the rabbit. It is bounded above by the quadrigeminal bodies, below by the nuclei of the vagus, laterally by the locus cæruleus and the nuclei of the auditory nerve. The greater part of this region is, therefore, situated in the brain territory we are now considering, and irritation occurring therein is very apt to occasion generalized epileptiform convulsions.

PARALYSES DUE TO LESIONS OF THE CEREBELLUM.

This subject is one surrounded by difficulty and uncertainty, partly dependent upon the obscurity that still prevails concerning the exact functions of the cerebellum, and partly because of the situation and anatomical connections of this organ, which are such as almost necessarily to entail a more than usual amount of functional disturbance of other important and related parts (medulla, pons, and sometimes of central ganglia, if there be impeded return of blood therefrom) when lesions or new growths occur within the substance of the cerebellum. Thus it happens that the 'indirect' symptoms due to lesions in the cerebellum, are often numerous, variable, and obtrusive—generally masking the 'direct' symptoms, which are themselves mostly vague and ill-defined in their nature.

Whilst it seems clear that the cerebellum has no independent function either in the direction of motility or of mental activity, we may also pretty safely affirm that it is much more intimately concerned with the production of bodily movements than with the evolution of mental phenomena.

The anatomical distinctness of the cerebellum from the larger brain and other parts of the nervous system is more apparent than real. Its roots, or 'peduncles,' penetrate into the cerebral hemispheres, into the pons, and also into the spinal cord through the medium of the medulla oblongata. The cerebellum is, as it were, an outgrowth from these several parts, and just as there is an anatomical interblending between it and them, so must there be a community of action in the bringing about of certain physiological results.

That there is an habitual community of action between the cerebellum and the spinal cord is certain; and the fact that an intimate functional relationship exists between the cerebrum and the cerebellum is shown by the circumstance that atrophy of one cerebral hemisphere (especially when it occurs in early life) entails a sequential atrophy of the opposite lateral lobe of the cerebellum. The consideration of certain well known facts which need not here be specified (see 'The Brain as an Organ of Mind,' p. 507), make it plain that the action of the cerebrum is primary, while that of the cerebellum is secondary or subordinate, in the performance of those functions in which they are both concerned.

If, however, the cerebellum acts as an abettor of the cerebrum, and the two together act upon, or through, the spinal cord, we ought to find that injuries of the cerebellum—in the absence of all disturbing causes—display themselves not so much by the lack of certain powers or faculties as by some diminution in the perfection with which they are exercised. Partial effects of this kind may, however, easily escape detection when slightly marked; and the precise amount of such defect in any given case can only be estimated with difficulty.

This, then, is one cause for the difficulty that has been so long felt in arriving at any definite and certain knowledge concerning the functions of the cerebellum. Another reason is to be found in the fact that the situation and connections of the organ are very unfavourable for the elucidation of its functions by means of experiments upon the lower animals. The loss of blood and shock from the operation are mostly severe before the cerebellum can be uncovered. And when to this disturbing cause, we add the fact that no attempt can be made to remove the organ without irritating most excitable and important parts of the nervous system to which it is adjacent, it must be clear that the difficulties and risks besetting this mode of investigation are extreme. The animals experimented upon have generally died before they have recovered from the shock of the operation, or, if not quite so soon, at all events before the effects of irritation of adjacent parts have had time to subside. One exception to this common order of events has of late been recorded by Luciani, and reference has been made in another section (p. 226) to certain important facts which his observations have made known.

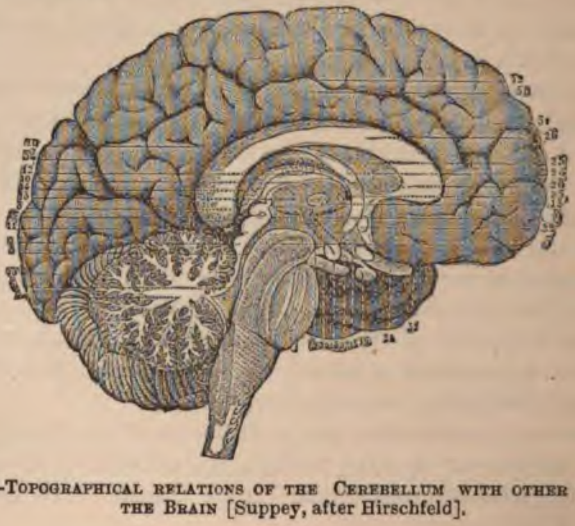

FIG 78.—TOPOGRAPHICAL RELATIONS OF THE CEREBELLUM WITH OTHER PARTS OF THE BRAIN [Suppey, after Hirschfeld].

A third reason for the backward state of our knowledge both concerning the functions of the cerebellum, and the signs and symptoms produced by disease in its substance, is to be found in the fact that the experiments naturally made by disease of this organ also speak to us in a vague or else in a confused manner, owing to the frequent contemporaneous interference with the functions of other important anatomically related parts. Shut in, as it is, within the narrow limits formed by the comparatively unyielding tentorium and the occipital bone, in company with the pons and bulb, injuries of the cerebellum are especially apt to compress or irritate one or other of these parts—to say nothing of additional disturbing influences which may be exerted in the cerebral hemispheres through the fibres of the superior cerebellar peduncles.

Another cause of complication is, moreover, apt to present itself in some cases. Alterations in the bulk of the cerebellum (and especially tumours in its middle lobe) may occasion more or less impediment to the proper emptying of the contents of the veins of Galen into the straight sinus, and may thus disturb the nutrition and functional activity of those important central ganglionic structures of the brain whose venous outflow takes this direction. Subsequently, in such a case, the functions of the brain will be still more widely interfered with, when well-marked dropsy of the ventricles has become established.

The different modes of operation of these disturbing influences are, of course, very various in individual cases of cerebellar disease, owing to the different single or combined causes of complication that may chance to come into operation in this or that case. Hence it is that we have such very considerable diversity in the symptoms presented by different cases of disease of the cerebellum. For the same reasons, we are often unable to say at all positively which of the symptoms are referable directly to destruction or to irritation of the cerebellar substance, and which to secondary or indirect effects of the lesions in question upon adjacent or related parts. We cannot positively decide this point even with reference to the hemiplegia which so frequently occurs in association with lesions in one lateral lobe of the cerebellum. But although some uncertainty still exists, the balance of evidence points strongly to the conclusion that the more or less complete paralysis of the opposite side of the body often met with in these cases is an 'indirect' result of injury to one lateral lobe of the cerebellum—an effect, that is, really induced by the pressure which such lesion occasions upon the corresponding anterior pyramid of the bulb.

This interpretation receives strong support from the fact that the hemiplegia, where present, habitually occurs on the side of the body opposite to that on which the lesion exists, although we have the best reasons for feeling assured that the functional relations of each lobe of the cerebellum is chiefly with the half of the cord, and consequently with the limbs, on the corresponding side of the body. This latter relation is obvious from the fact that atrophy of one hemisphere of the cerebellum leads to atrophy of the opposite lateral lobe of the cerebellum and also to some atrophy of the opposite half of the spinal cord in its lateral regions. We are entitled to conclude, therefore, that all direct effects resulting from lesions in one lateral lobe of the cerebellum would (so far as they are concerned with motility) manifest themselves principally upon the side of the body corresponding with the lesion. This, of course, is a conclusion of some importance.

On the other hand, it is even more certain that although afferent impressions of all kinds are conducted towards the cerebellum (principally by way of its inferior peduncles), yet lesions of this organ do not, by destroying its tissue, cause any interference whatever with the general or special sensibility of the body. The reason for this seems clear. Such afferent impressions as are conducted to the cerebellum do not directly evoke mental phenomena or in any way reveal themselves in consciousness; they act as unconscious incitors of this or that group of muscular movements, capable of being initiated and regulated by a strictly corresponding and coordinate outflow of cerebellar energy. In other words, they act as ingoing impressions which are related to sets of movements produced under the guidance of the cerebellum, in just the same sort of way that other ingoing impressions are related to sets of movements (reflex), produced under the guidance of the spinal cord. In making use of this explanation I do not wish to say that the analogy can be pushed too far, or to express an opinion as to the extent to which in the performance of movements the cerebellum can be called into action without the concurrent activity of the cerebrum, but merely to point out that an injury to the cerebellum which should chance to involve mainly such afferent fibres, would cause no diminution primarily in the sphere of consciousness (or in other words, no anæsthesia of any part of the body), and would produce only some amount of interference with the individual's power of evoking certain movements.

How far diminished mental energy may, like diminished muscular energy be an occasional marked result of disease of the cerebellum is a moot point; still it seems possible that some such result is at times rightly to be ascribed to this cause (Luys). We call attention to this point in order that future observers may endeavour to throw light thereon.

For purposes of diagnosis we have to consider the existence or absence of certain groups of symptoms, and for the most part, as we have previously had occasion to remark, irrespective of the question whether this or that sympton belongs to the 'direct' or the 'indirect' category.

Facts of importance concerning the blood supply of the Cerebellum. —Very large hæmorrhages are occasionally met with in the cerebellum, owing as Duret pointed out to the rupture, on either side, of a large branch of the superior cerebellar artery which supplies the grey nucleus (corpus rhomboidale) by its hilum, there breaking up into radiating branches for the nourishment of this organ and of the white substance around. Rupture of this branch may produce an extravasation of blood so large as to occupy nearly the whole of the corresponding lobe, or more rarely still nearly the whole of the cerebellum. A rupture of one of the radiating offsets of this branch, however, for the most part gives rise to a small extravasation contained within the rhomboidal body itself.

A hæmorrhage into the more superficial portions of the cerebellum may partly involve white matter alone, and partly the grey matter of contiguous leaflets. Such hæmorrhages are comparatively unimportant owing to their small size—commonly varying between that of a pea and a small almond in size.

The *superior* and *inferior peduncles* of the cerebellum only contain vessels a little larger than capillaries, and are very rarely the seats of hæmorrhage. The *middle peduncle* is supplied with larger vessels, derived almost exclusively from the middle cerebellar artery, and here larger extravasations of blood are also occasionally met with, which, following a longitudinal direction, separate the fibres of the peduncle and may reach as far as the pons.

The three sets of vessels that ramify over the surface of the cerebellum usually anastomose with one another and with their fellows on the opposite side. Occlusion of different groups of these vessels occurs from time to time (from thrombosis, rarely from embolism) and may thus give rise to foci of softening which are extremely variable in site and size, and often very ill defined in their boundaries. An obliteration of the vessel supplying the corpus rhomboidale on one side, leading to softening of that body, ought to produce an extremely important grouping of symptoms (as yet unrecorded) if it were to occur alone. Such softenings, however, are only too apt to be complicated by others in remaining territories of the superior cerebellar artery, as well as in areas supplied by the trunk of the posterior cerebral artery from which it is derived, and thus no clear light would be thrown by such cases upon the problems of regional diagnosis in cerebellar disease.

Signs and Symptoms of Lesions in Different Parts of the Cerebellum.

1. *Lesions in the Lateral Lobe of the Cerebellum.* Lesions may occur in this region with or without loss of consciousness, according as they are sudden and extensive or the reverse.

Severe pain in the occiput, or it may be in the frontal region, is often complained of—mostly paroxysmal in character. Vomiting occurs perhaps rather more frequently than with lesions in other parts of the brain.

Paralysis of the opposite arm and leg, usually incomplete, is very often met with, though it is not commonly accompanied by much diminution of sensibility. The paralysis is apt to be more marked in the leg than in the arm, and it is also generally absent from the face. The condition differs, therefore, in both these respects from the more ordinary forms of hemiplegia, though such a combination of paralysis may likewise be produced by certain limited lesions of the cortex in the parietal region (p. 281), and possibly also by lesions in particular parts of the internal capsule.

In the rare cases in which the face is paralysed, it may be on the side of lesion (brought about by pressure on the facial nerve), and the upper as well as the lower part of the face is then found to be affected.

There may or may not be some early rigidity in the paralysed limbs; perhaps, also, some trismus, spasms about the face or of the ocular muscles, or marked rigidity of the muscles of the back of the neck (with drawing back of the head).

There is often a tendency to fall or deviate towards the side of lesion, in walking.

Speech is generally not affected, and the movements of the tongue are mostly unimpeded. In the majority of cases also, deglutition is not interfered with; but in certain cases where there is well-marked pressure upon the bulb, there may be dysphagia as well as some difficulty in articulation.

No definite observations have, I believe, as yet been made in cases of this kind, as to the relative temperature of the two sides of the body.

Blindness may supervene in some cases, but principally in association with tumours in this region, and as a sequence of optic neuritis, when this condition gives place to one of atrophy.

Throughout, there is usually no delirium or mental impairment. At the most there may be some intellectual slowness or dulness; or perhaps more or less drowsiness, when there is much increase of intra-cranial pressure. Towards the close, in fatal cases, coma often supervenes, owing to an extension of the lesion or to some independent mischief.

At times the symptoms resulting from lesions in one lateral lobe of the cerebellum are still less definite. There may be no trace of a hemiplegic condition, but rather a general muscular weakness of a progressive character, associated with a peculiar, unsteady, and vacillating gait (titubation). This gait is more fully described in the next section, as it most frequently exists to a marked degree with disease in the middle lobe of the cerebellum. The paresis is commonly more marked in the legs than in the arms. Sometimes the amount of general weakness and prostration has been extreme, so that the patient has been quite unable to stand or even to raise himself in bed.

2. *Lesions in the Median Lobe of the Cerebellum.* When a lesion exists in this part of the cerebellum, the symptoms produced are also apt to vary widely—the differences being due for the most part to differences in the extent and degree of abruptness of the lesion. As a rule, the lesions of the most serious type existing in this situation are chronic in their mode of onset—consisting principally of new formations. Anything like a large hæmorrhage or an extensive softening limited to this region is excessively rare; smaller lesion of this type may be met with in the middle lobe alone, but they give rise to much less grave and definite symptoms. A large hæmorrhage having its origin in one of the lateral lobes may of course extend so as to involve the middle lobe; and so, occasionally, we may have a softening which involves simultaneously the middle and one of the lateral lobes. In these latter cases the grouping of symptoms is correspondingly mixed. Thus it happens that typical uncomplicated

disease of the middle lobe of the brain when it can be diagnosed carries with it a strong presumption that the disease is a tumour—the establishment of the regional diagnosis goes far, indeed, to carry with it a pathological diagnosis.

Signs of hemiplegia are, as a rule, absent when we have to do with disease of the middle lobe of the cerebellum, though there is general and increasing paresis, associated with the peculiar waddling or ataxic gait (titubation) well known to be not unfrequently connected with cerebellar disease. The gait has been compared to that of a drunken man, but in some cases it has seemed to me closely to resemble that of a person who walks on the deck of a vessel in a rather rough sea. The legs are wide apart and the progression is staggering and uncertain, without the spasmodic movements or the bringing of the heel down with a stamp, such as we look for in posterior spinal sclerosis. In cerebellar ataxy there is the further important characteristic, that, when standing still, closure of the patient's eyes causes no increase of unsteadiness or tendency to reel, as it does in posterior spinal sclerosis. Again, in the cerebellar affection there is probably no impairment of sensibility of any kind—the exceptions to this rule being very rare.

There may be rigidities of the posterior cervical muscles and also, occasionally, of one or more of the limbs. There may also be convulsive attacks from time to time in which tetanic spasms are in excess, with retraction of the head and arching of the body (H. Jackson).

The general signs of tumour are often extremely well-marked. Sickness is more than commonly frequent and severe; headache, occipital in site, is often intense, though at other times it is felt most in the frontal region; optic neuritis, again, is often severe and not unfrequently leads to optic atrophy and amaurosis.

Amaurosis may also possibly be induced in another way. The corpora quadrigemina and geniculate bodies may be actually invaded by new growths involving the middle lobe of the cerebellum.

Lesions of the middle lobe of the cerebellum are the forms of disease of this organ, in which, if it occurs at all, excitation of the genital organs is met with—not so much perhaps from the disease in the cerebellar tissue, as owing to the irritation which such disease may set up in the posterior aspect of the bulb. Symptoms of this type have been met with in about one-third of the recorded cases of disease of the median lobe of the cerebellum. Increase of sexual desires may be experienced, and in male patients there have been frequent erections with or without emission. No such symptoms are caused by lesions in the lateral lobes.

Another symptom occasionally met with in disease of the middle lobe of the cerebellum, is a tendency to fall forwards where the lower part of this segment is specially implicated; and, on the other hand, a tendency to fall backwards when the upper part of the middle lobe is involved. Sometimes these signs are well marked.

Varied and peculiar oscillatory movements of the eyeballs allied to those of nystagmus are sometimes encountered in association with lesions of the middle lobe of the cerebellum. They may be vertical, horizontal, or oblique and are generally parallel (Mackenzie). Sometimes these movements are observed only during convulsive attacks; but at other times (and especially in association with rigidities in the cervical or other muscles), the eyes may be fixed in one direction, or they may present slight parallel oscillatory movements.

Tumours in the middle lobe of the cerebellum are especially apt to lead to ventricular dropsy and perhaps a certain amount of central softening, by causing more or less impediment to the return of blood from the veins of Galen into the straight sinus, as well as (according to Mackenzie) from obliteration of the communication between the third and fourth ventricles and the consequent inability of ventricular fluid to escape into the great subarachnoid spaces. Where such effects are induced, and especially when the patient is a child, chronic hydrocephalus is set up and the head soon begins to enlarge. No absolutely certain test of the supervention of this complication is to be found, in such a case, other than the establishment of an actual increase in the dimensions of the head; hence in all instances in which a diagnosis of tumour of the middle lobe of the cerebellum is made, it is important carefully to measure the circumference and breadth of the head at once, in order to obtain data that may be of use, subsequently, in determining whether chronic hydrocephalus is being established or not. Of course the gravity of the symptoms would be distinctly increased by the springing up of this complication; but, apart from the physical sign above mentioned, not in a distinctive manner—that is, not in ways which might not be caused by mere increase of the original disease and a consequent augmentation of intra-cranial pressure.

3. *Lesions in the Middle Peduncle of the Cerebellum.* With

lesions in this situation (hæmorrhages, abscesses, or tumours), there is a tendency similar to that which has been observed in animals in whom the middle cerebellar peduncle has been cut, for rotation to occur round the longitudinal axis of the body. The direction of rotation is commonly from the sound towards the injured or diseased side; occasionally, for some not fully explained reason, the rotation has been noted in the opposite direction. In the case of a patient in a partial apoplectic state, or condition of stupor, this tendency may manifest itself only, by his lying on the injured side with the head strongly turned in the same direction.

This tendency to rotation round the longitudinal axis of the body is commonly associated with a remarkable divergence of the eyes. The eye on the injured or diseased side is directed downwards and inwards, whilst that of the sound side is turned as strongly upwards and outwards. They may be motionless in these directions, or may exhibit a certain amount of nystagmus (Nonat).

Similar signs may be met with where the root of the peduncle is implicated in the substance of the lateral lobe of the cerebellum. And in these cases, generally, if any paresis is produced, it should be looked for on the same and not on the opposite side of the body. The phenomena of rotation and the paresis are best explained (Vulpian) by the supposition that there is a cutting off of some motor stimulus from the muscles of the side of the body corresponding with the lesion (with which the corresponding lobe of the cerebellum is in relation), and consequently an unbalanced action of the muscles of the sound side of the body.

There is no special tendency for isolated lesions to occur either in the *Upper* or in the *Lower Peduncles* of the cerebellum, nor can anything definite be said concerning the symptomatology of lesions limited to either of these situations.

PART II.

PARALYSES OF BULBAR ORIGIN.

PRELIMINARY REMARKS.

Although the Bulb or Medulla Oblongata is commonly regarded as a constituent part of the brain, yet there are many practical reasons which make it expedient to consider the morbid conditions of this segment of the cerebro-spinal system in a section altogether separate. Though as a part contained within the cranium it is rightly enough regarded as a component of the brain, yet it is no less true that it is the direct morphological continuation of the spinal cord, and we might, on this account, with equal justice, refer to its morbid conditions in connection with those of this segment of the nervous system.

But reasons against either of these courses, and in favour of dealing with the morbid conditions of the bulb in a section altogether apart, are easily found. On the whole, and especially from the point of view of the mode in which morbid conditions affect it, the bulb is certainly more closely related to the spinal cord than it is to the brain; on the other hand, the mere fact that the bulb is contained within the cranial cavity, causes it to be involved not unfrequently in a secondary manner in various cases of brain disease—that is, by the extension of some lesion originating in a contiguous part of the brain, or by undue pressure upon the bulb caused by certain intra-cranial lesions (Fig. 77). Thus, although, in regard to its own independent diseases, the bulb is more closely related to the spinal cord, it is, as a matter of fact, frequently involved in common with some part of the brain. The great intrinsic importance of disease in this particular region of the cerebro-spinal system affords another good reason for the separate consideration of its morbid conditions.

SECONDARY AFFECTIONS OF THE BULB.

The information that will subsequently be given, in reference to the signs and symptoms of disease of the bulb, must be made use of by the practitioner in order to enable him to diagnose the secondary or coincident involvement of this part, in cases where disease exists in other encephalic regions. This secondary implication of the bulb, occurs more especially when the pons is the seat of hæmorrhage, softening, or new growth; or when similar morbid processes in some part of the cerebellum produce so much pressure upon the bulb as distinctly to interfere with some of its functions; or, finally, when disease in some other part of the brain exists of such a nature as to lead to a great excess of intra-cranial pressure, which tells upon the functions of the medulla as well as upon those of other parts. In some of these latter cases, as in large intraventricular hæmorrhages when blood passes from the third into the fourth ventricle, the bulb may be directly compressed, and death is apt to be speedily brought about.

It must, of course, be fully understood that in other cases, disease-processes become communicated to the bulb from the spinal cord, as in Landry's paralysis, in amyotrophic lateral sclerosis, etc.; and that, in other instances still, morbid changes may spring up coincidently in the bulb and in the spinal cord; or even in the cerebrum, the bulb, and the spinal cord—as in certain cases of disseminated cerebro-spinal sclerosis.

PRIMARY AFFECTIONS OF THE BULB.

Here we shall deal only with morbid processes that originate in the bulb itself, and with the morbid signs and symptoms to which they give rise.

The anatomical limits of the bulb correspond with the decussation of the pyramids and roots of the first cervical nerve fibres below, and with the lower border of the pons above; while, posteriorly, its upper boundary will be the upper striæ medullares which cross the floor of the fourth ventricle. The upper half of the floor of this ventricle we have, for convenience sake, described as part of the pons —a proceeding also warranted by the fact that the grey nuclei included in this upper part of the fourth ventricle are supplied by the median vessels of the pons. Erb has adopted the opposite course, and described the whole of the floor of the fourth ventricle as

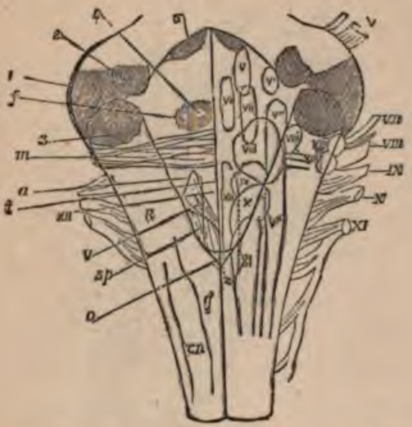

FIG. 79. DIAGRAMATIC VIEW OF THE POSTERIOR SURFACE OF THE MEDULLA, OR FLOOR OF THE FOURTH VENTRICLE, SHOWING THE RELATIVE POSITIONS OF DIFFERENT NERVE NUCLEI [after Erb].

The left half of the figure represents:—Cn, funiculus cuneatus, and *g*, funiculus gracilis; O, Obex; *sp*, nucleus of the spinal accessory; *p*, nucleus of the pneumogastric; R, restiform body; XII′, nucleus of the hypoglossal; *t*, funiculus teres; *a*, nucleus of the acousticus; *m*, striæ medullares; 1, 2, 3, middle, superior and inferior cerebellar peduncles. *f*, fovea anterior; 4, eminentia teres (genu nervi facialis); 5, locus cœruleus.

The right half of the figure represents the nerve nuclei diagrammatically:—V, motor trigeminal nucleus; V′, median, and V″, inferior sensory trigeminal nuclei; VI, nucleus of abducens; VII, facial nucleus; VIII, posterior median acoustic nucleus; VIII′, anterior median; VIII″, posterior lateral; VIII‴, anterior lateral acoustic nuclei; IX, glosso-pharyngeal nucleus; X, XI, XII, nuclei of vagus, spinal accessory, and hypoglossal nerves respectively. The Roman numerals at the side of the figure from V to XI represent the corresponding nerve roots.

a part of the bulb. But for mere diagnostic purposes, it would seem that the course here adopted is the best.

We shall elsewhere, in part, and in an incidental way, have to consider this same subject. That is, in dealing with the diagnostic aspect of morbid conditions affecting the different Cranial Nerves, we shall have to refer to bulbar affections in which the nuclei of one or other of these nerves are involved. A reference to Figs. 79, 80 will show that some of the nuclei of the cranial nerves are situated only in part within the confines of the bulb—such as the inferior sensory nucleus of the trigeminus, the nucleus of the auditory, a lower facial nucleus, and the nucleus of the spinal accessory; while, on the other hand, some of them are situated wholly within the confines of the bulb, such as the nuclei of the glosso pharyngeal, of the pneumogastric, and of the hypoglossal. For an account of the full effects

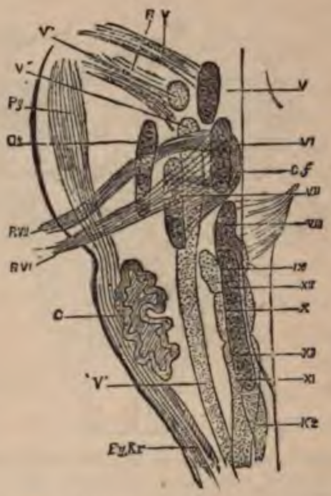

FIG. 80. TRANSPARENT LATERAL VIEW OF THE MEDULLA, SHOWING DIAGRAMATICALLY THE RELATIVE POSITIONS OF THE MOST IMPORTANT NERVE NUCLEI [after Erb].

The right half of the Medulla seen from the surface of section; the parts that lie closer to this surface are most deeply shaded.

Py, pyramidal tract; Py, Kr, decussation of pyramids. O, olivary body; Os, superior olivary body; V, motor, V/, middle sensory, V, inferior sensory nucleus of trigeminus; VI, nucleus of abducens; Gf, genu facialis nervi; VII, nucleus facialis; VIII, posterior median acoustic nucleus; IX, glosso pharyngeal nucleus; X, nucleus of Vagus; XI, nucleus of the accessorius; XII, hypoglossal nucleus; Kz, nucleus of the funiculus gracilis; Rv, trigeminus root; RVI, root of the abducens; RVII, root of the facialis.

ensuing from the paralysis of these several nerves reference must be made to the subsequent part (Part III.) of this work.

PATHOLOGICAL DIAGNOSIS.

GENERAL CONSIDERATIONS CONCERNING THE SEVERAL CONDITIONS WHICH CAUSE PARALYSES OF BULBAR ORIGIN.

We here enumerate, and shall have something to say concerning, the several causes of paralyses of bulbar origin, though it is difficult to state in any precise way their order of numerical frequency.

A.—TRAUMATIC CAUSES.

B.—RUPTURES OF BLOODVESSELS WITHIN THE SUBSTANCE OF THE BULB.

C.—OCCLUSIONS OF BLOODVESSELS SUPPLYING THE BULB (leading to foci of Softening).

D.—CHRONIC MENINGITIS.

E.—TUMOURS ORIGINATING IN THE BULB OR ITS MENINGES.

F.—DISSEMINATED SCLEROSIS.

G.—DEGENERATIVE CHANGES IN MOTOR GANGLION CELLS (Labio-glosso-laryngeal paralysis).

The changes we have mentioned above are those most likely to occur as actual organic causes of paralyses of bulbar origin.

Cases are, of course, by no means unfrequently met with in which there is a marked impairment of this or that function of the medulla oblongata, or of some small groups of such functions, in which from the shifting nature of the signs and symptoms, and the fitfulness of their manifestation, together with other peculiarities, we are compelled to come to the conclusion that they are due to functional rather than to structural disease of the bulb. We generally come to such a diagnosis here, as in other similar cases, by way of exclusion—we arrive at the opinion, that is, that the sum-total of signs and symptoms presented, their variability, together with the history of the patient generally, is not compatible with the notion that the bulb can be the seat of either one of the recognizable morbid changes above enumerated.

A loose nomenclature has obtained considerable currency during recent years, seeing that one of the several forms of paralysis of bulbar origin (labio-glosso-laryngeal paralysis), which, for the sake of brevity, was named by Wachsmuth 'progressive bulbar paralysis,' is only too frequently referred to in this country merely as 'bulbar paralysis'—which is an absurdly exclusive designation for this disease, seeing that instead of one, there are many different forms or combinations of bulbar paralysis due to organic changes of this or that variety.

A. TRAUMATISMS.

Severe blows on the head causing sudden death are at times associated with contusion of, or punctiform hæmorrhages into, the bulb (Duret and Westphall). In some other cases where blows on the head have been received, which have not proved fatal, diabetes or polyuria has, after a time, resulted. These conditions may have been due to changes of some kind in the fourth ventricle, caused or initiated by the traumatism.

In some cases of falls upon the head, or in the case of sudden twists or strains of the head and neck, there may be fracture or dislocation of the atlas and of the axis, attended by a crushing pressure upon the bulb, leading to instant death owing to the arrest of the circulation and respiration produced by shock or damage to the corresponding centres. In cases where disease of these vertebræ has pre-existed, such fractures or dislocations have occasionally been brought about by the most trivial strains or blows (Ollivier).

Crushing lesions of the medulla are commonly due to one or other of the following causes:—dislocation of the cranium from the atlas, or of the atlas from the axis (the odontoid process slipping out from its ligament); or from fracture of the occipital bone (with displacement), or fracture of the odontoid process.

B. RUPTURES OF BLOODVESSELS WITHIN THE BULB.

Primary hæmorrhages into or upon the surface of the bulb are decidedly rare events when compared with the comparative frequency with which such a pathological accident occurs within the pons and other parts of the cerebral hemispheres. Hæmorrhages into the spinal cord are also rare, though perhaps not so rare as they are within the bulb.

In some cases an extravasation commencing in the pons may extend thence into the contiguous portion of the bulb, and the result of such a lesion is likely to be speedy death. Only a very few cases of primary hæmorrhage into the medulla have been recorded; and though such extravasations have nearly always been very small, not much larger than a pea, they have yet given rise to extremely serious symptoms. They are most apt to occur in the region of the bulb contiguous to the pons, during severe straining efforts, when the person is suffering from some violent emotion, when he is in a state of alcoholic intoxication, or under the influence of any other condition which greatly increases the tension upon the walls of the bloodvessels. The part of the bloodvessel that actually gives way may be here, as in other regions of the brain, previously dilated into a small miliary aneurysm.

C. OCCLUSION OF BLOODVESSELS WITHIN THE BULB LEADING TO FOCI OF SOFTENING.

A reference to Figs. 70 and 72 will show that the bulb derives its blood supply exclusively from the vertebral arteries and will make plain the manner in which the branches of these vessels are distributed over its surface and through its substance.

Of the two causes of vascular occlusion, *Embolism* is unquestionably the rarer of the two. Still, embolism of one of the vertebral arteries occurs at times, or we may have smaller emboli driven into one or more of the branches of either of these vessels. The symptoms produced will, of course, vary much according as the trunk of the artery or this or that branch becomes occluded; though in all cases they will, like those caused by hæmorrhages, be distinguished by their abrupt mode of onset. Owing to its mode of origin from the subclavian, embolism is met with more frequently in the left than in the right vertebral artery or its branches.

Thrombosis in either vertebral artery, or in this or that branch, occurs with decidedly greater frequency than embolism. The symptoms are equally variable according to the site of the occlusion; but are generally developed in the course of hours or days, rather than abruptly. At times the actual onset of some symptoms is abrupt, though the condition goes on for a while increasing in severity.

D. CHRONIC MENINGITIS.

Chronic meningitis is most likely to be of syphilitic origin, and may or may not be associated with disease of contiguous parts of the occipital bone. Where such a condition exists, symptoms are most likely to be caused by the involvement of the roots of some of the nerves connected with the surface of the medulla—viz., of the sixth, the seventh, the auditory, the glosso-pharyngeal, the vagus, the spinal accessory, or the hypoglossal. Some of these combinations are mentioned, and the effects of the involvement of this or that nerve are fully described, in the part of this work devoted to the consideration of lesions of the Cranial Nerves. Evidence of the involvement of the roots of the cranial nerves, is founded in the main, as there set forth, upon the particular combinations of paralysis encountered, which may be only explicable, or most easily explicable, on the supposition that disease exists in such a site. Still, multiple wide-spread lesions might produce like symptoms, in some cases, and such multiple lesions are very apt to occur in syphilis.

E. TUMOURS ORIGINATING IN THE BULB OR IN ITS MENINGES.

The bulb may be involved by tumours originating in contiguous parts, such as the pons or the cerebellum, and here a mixed symptomatology is of course produced.

In the case of tumours originating in the bulb or its membranes, with which we are now specially concerned, we must in any case be guided towards such a diagnosis, first by the general symptoms indicative of tumour, such as optic neuritis, cephalalgia, sickness; while for the regional diagnosis, we must rely upon the existence of such combinations of symptoms as are most apt to occur, and such as will be cited in the section devoted to this part of our subject. It is possible for a tumour to grow, even in such a situation as the bulb, provided it increases slowly and does not attain any great size, without producing very marked symptoms (Wilks).

F. DISSEMINATED OR INSULAR SCLEROSIS.

The bulb and the pons are parts that are specially prone to be implicated in disseminated sclerosis. Occasionally it happens, as in a case which I have elsewhere recorded (' Trans. of Clin. Soc.' 1884), that the first morbid changes of this type may develop in the bulb, and may for some time be limited to this region. The diagnosis would then rest, in the main, between such changes as we are now considering and the existence of a tumour. The latter was negatived and the diagnosis of disseminated sclerosis supported in the case referred to, by the fact of the absence of the general symptoms indicative of tumour (optic neuritis, headache, sickness), together with evidence of the existence of a lesion capable of dissecting out contiguous parts—affecting some and wholly sparing others, *e.g.*, affecting both auditory nerves or nuclei whilst sparing both facials.

In the large majority of cases of disseminated sclerosis, the bulb is affected in common with parts of the brain above, and parts of the spinal cord below, though the bulbar lesions give rise to some of the most characteristic symptoms.

G. DEGENERATIVE CHANGES IN MOTOR GANGLION CELLS OF THE BULB (LABIO-GLOSSO-LARYNGEAL PARALYSIS).

In this affection we have to do with changes in the medulla similar in kind, and homologous in site, with those which, when occurring in the spinal cord, give rise to progressive muscular atrophy. Some regard the changes in question as being of a chronic inflammatory order, others believe that they should rather be considered as degenerative changes of a peculiar type.

The principal nuclei that become affected by these changes (which soon eventuate in the production of marked atrophic processes in the ganglion cells) are that of the hypoglossal (leading to paralysis of the tongue muscles) and that of the accessory portion of the spinal accessory (leading to paralysis of laryngeal muscles). In these nuclei the changes above mentioned have been clearly recognized. But some doubt is entertained as to the precise part of the facial nucleus which is involved in this disease—or, in other words, as to the precise part or nucleus from which those nerve fibres arise that innervate the lip muscles. It was suspected by Lockhart Clarke to be a lower or accessory nucleus of the facial nerve, situated partly on the inner side and partly behind the hypoglossal nucleus (Fig. 81, *if*, *ef*,) ; this, though possibly correct, cannot be considered as settled at present. It is quite certain that the lips and the tongue are closely related in their movements ; and, therefore, it is by no means improbable that these portions of the facial nucleus, contiguous to that of the hypoglossal, may be the parts chiefly concerned with the movements of the lips. The muscles of the palate and pharynx are also affected in this disease. The question whether a motor division to the nucleus of the glosso-pharyngeal nerve exists, formerly held in doubt, seems now to be settled in the affirmative. This nucleus may become affected in labio-glosso-laryngeal paralysis, and from it some of the fibres supplying palatine and pharyngeal muscles may be derived, though it seems highly probable that other of the motor fibres which are distributed with the glosso-pharyngeal nerve really proceed from the spinal accessory nucleus, just like those motor fibres that are distributed with the vagus.

The relative position of the motor nuclei whose disease is thus supposed to be concerned with the production of this well marked and important disease may be gathered from the following figure.

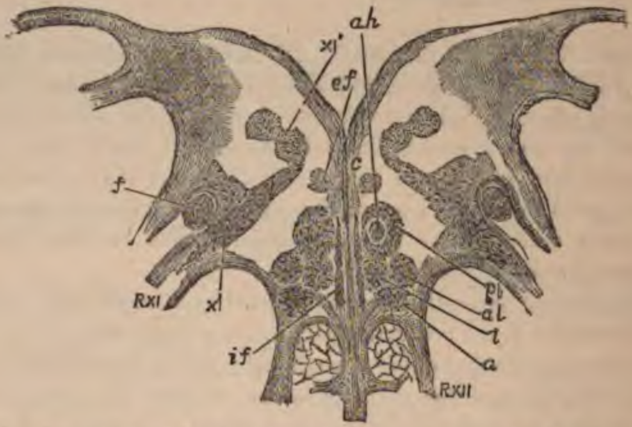

FIG. 81. SECTION OF THE BULB, A LITTLE BELOW THE POINT OF THE CALAMUS SCRIPTORIUS, SHOWING THE GROUPS OF CELLS OF THE GREY SUBSTANCE [after Young and Ross].

Rxi, Fibres of origin of the eleventh or Spinal Accessory Nerve.
xi, Posterior nucleus of the eleventh nerve.
xi', Accessory nucleus of the eleventh nerve.
Rxi, Fibres of origin of the twelfth or Hypoglossal Nerve.
a, *i*, *al*, *pl*, Anterior, internal, antero-lateral, and postero-lateral groups of cells respectively.
ah, Accessory hypoglossal nucleus.
if, Internal accessory facial nuclei.
ef, External accessory facial nucleus.
C, Central canal. *f*, Fasciculus rotundus.

The morbid affection of these nerve nuclei, viz., of the hypoglossal, of part of the facial, of the glosso-pharyngeal, and of the spinal accessory, are the primary events of the disease, and the actual causes of the different forms of paralysis by which the malady is characterized.

We require merely to mention here the fact that the nerve fibres arising from these nuclei become atrophied, and that there is a consequent tendency in the muscles to which the nerve fibres are distributed to undergo atrophy and degeneration. It so happens, however, that this atrophy becomes appreciable to us during life, if at all, only in the muscles of the tongue, or more rarely still in the lips; although it may exist, it escapes notice in the palate, throat and larynx. But it is only in a certain proportion of the cases that distinct diminution in bulk is to be made out in the tongue on one or both sides, and then it is generally associated with fibrillary twitchings. In other cases, although atrophy of the paralysed muscular fibres of the tongue occurs, the loss of bulk is compensated by an overgrowth of connective tissue and fat between the atrophied fibres. The organ presents, of course, a very different appearance in the two sets of cases; in the former, it is shrivelled and more or less wrinkled; while in the latter, it is large, flabby, and smooth on the surface.

As further elements in aid of diagnosis, it should be stated that this disease occurs principally in elderly persons—rarely in those under forty—and that, in nearly one half of the cases, it is found to have supervened in a person who has already been affected with progressive muscular atrophy involving the muscles about the shoulder, or those of other parts of the upper extremities. It is a rare event for labio-glosso-laryngeal paralysis to occur first, and to be followed by signs of progressive muscular atrophy in other parts of the body. It is however by no means rare for labio-glosso-laryngeal paralysis to exist alone up to the end; in the same way that progressive muscular atrophy, dependent upon lesions in this or that part of the spinal cord, may exist alone, without evidence of any implication of the motor nuclei in the bulb.

These relations between the two diseases will be readily understood, if we bear in mind the fact that they are both of them dependent upon essentially the same kind of morbid process—the two diseases being merely different manifestations, consequent upon the fact that the bulb is involved in labio-glosso-laryngeal paralysis, while different portions of the spinal cord are affected in progressive muscular atrophy. The morbid process may occur in either part of the cerebro-spinal system separately; or it may occur first in one of these regions, and subsequently show evidence of its existence in the other.

The combination of symptoms is often so distinct in this slowly progressive disease, that the recognition of its nature, from a regional point of view, enables us to decide at the same time as to the nature of the morbid process which underlies it—or, in other words, the establishment of the regional diagnosis, carries with it the pathological diagnosis. Precisely the same thing may be said concerning progressive muscular atrophy as an affection of the spinal cord. When the two conditions coexist, the diagnosis should be made with equal readiness.

PATHOLOGICAL DIAGNOSIS.

CLINICAL INDICATIONS FAVOURING THE EXISTENCE OF THIS OR THAT CAUSATIVE CONDITION.

The causes of paralyses of bulbar origin just referred to are divisible into two principal categories if we look to the degree of rapidity with which, in different cases, the signs and symptoms of the several diseases are developed. Some give rise to *acute*, and others to *chronic* disease of the bulb.

CAUSES OF ACUTE BULBAR DISEASE.	CAUSES OF CHRONIC BULBAR DISEASE.
Traumatisms.	Chronic Meningitis.
Hæmorrhages into the substance of the Bulb.	Tumours in the Bulb and in its Meninges.
Embolic occlusion of arteries.	Disseminated sclerosis.
Thrombotic occlusion of arteries.	Degenerative changes in motor ganglion cells (labio-glosso-laryngeal paralysis).

Where **traumatisms** have occurred as the causes of symptoms of bulbar paralysis, the symptoms are of absolutely sudden onset, and we have for our guidance either the history of some severe blow or fall, or else evidence of the same presented by the patient's condition, or by the place and position in which he was found. In another class of cases, in which dislocation or fracture of the atlas or of the axis has occurred, as a result of caries or other disease of these vertebræ, the existence of the vertebral disease has generally been previously known or at least suspected.

In the very rare cases in which **hæmorrhage** into the bulb occurs as a primary event, the onset of symptoms is also abrupt, and this abrupt onset becomes all the more significant when it shows itself in an elderly person, or even in a person over forty years of age. After this time of life, even should a cardiac bruit exist, embolism is an extremely rare event, since the cause of any coexisting cardiac bruit is likely to be mere degenerative thickenings of the valves, rather than a plastic endocarditis capable of furnishing embolic fragments.

Even a small bleeding into the bulb, when the blood is poured out quickly into such a site as the nucleus of the vagus, may cause sudden death by arresting respiration or cardiac action. In other cases, though the symptoms may be very grave at first, the patient may, after hours or days, recover from this primary condition of shock, and be left with this or that set of symptoms indicative of a bulbar lesion, such as will be noticed when speaking of the regional diagnosis. There may be produced in this way an acute form of labio-glosso-laryngeal paralysis, with or without the association of paresis of one or more limbs, but differing further from the ordinary chronic affection by reason of its non-progressive character.

Embolic occlusions of vessels supplying the bulb are also decidedly rare. For some indications of diagnostic importance in reference to embolism of cerebral vessels generally, reference should be made to what has previously been said at p. 33. It is only necessary to repeat here, as of primary importance for such a diagnosis, the abrupt appearance of cerebral symptoms indicative of implication of the bulb, in a child or young person who is the subject of valvular disease. It has been already mentioned (p. 357) that the mode of origin of the left vertebral from the subclavian favours embolism in the territory of this artery rather than in that of its fellow. It is, further, worthy of note that whether we have to do with embolism of the main trunk of the medullary portion of the vertebral (an extremely rare event), or with embolism of one or other of its branches, we should in either of these cases look for unilateral symptoms—since it would be an event in the highest degree improbable that embolism should occur simultaneously in the territories of both vertebral arteries, except in the very rare cases of multiple cerebral embolism, in which the symptoms referable to a bulbar lesion would, perhaps, be merged by the existence of an apoplectic state or otherwise obscured.

Embolism from malignant or septic endocarditis may, it is true, occur in persons of any age, simply because this disease is not like plastic endocarditis confined to the first half of life and especially to its earlier decades. Here, however, the symptoms of the general disease with its multiple manifestations would go far to obscure the diagnosis and render embolism of bulbar arteries (should it occur) difficult or impossible to recognize.

Thrombotic occlusions of vessels supplying the bulb are decidedly more common than those due to embolism ; they are also more common than primary hæmorrhages in this situation. What has been said elsewhere concerning the periods of life and conditions under which thrombosis is most prone to occur in cerebral vessels generally (p. 30), holds good for the vessels of the bulb. It is only needful to say here that no age is exempt from the liability to such changes, although they are most frequent in the latter half of life—just at the time, that is, when hæmorrhages are also most likely to occur.

In cases where the diagnosis lies between thrombosis and hæmorrhage, it may be said that, other things equal, the younger the patient, the more is the chance that we have to do with thrombosis—especially if the patient is suffering from, or has recently recovered from, some acute specific disease. Again, as regards the mode of onset, in the case of thrombosis it is much more apt to be preceded by some premonitory symptoms ; or if the actual onset of serious symptoms should be abrupt in the case of thrombosis (as sometimes happens), the symptoms are then more prone to go on developing for a time than when we have to do with a case of hæmorrhage.

The trunk itself of one of the vertebrals is occasionally the seat of the thrombosis, but even here the symptoms may vary much in different cases according to the situation in which the thrombus exists, and whether or not it cuts off the blood supply from the anterior spinal, or from the inferior cerebellar artery, or from both. It has happened in some recorded cases that first one and then the other vertebral artery has become thrombosed, with the effect of ultimately producing symptoms similar to those due to thrombosis of the basilar artery (Duffin).

Where some small branch only becomes affected, the symptoms will, of course, vary widely according to the nature of the vessel and the particular nuclei which it supplies. Occasionally minute foci of softening may be found scattered through the medulla, pons, and other parts of the brain. This was the case in a patient recently under my care, in which there was reason to believe that the medulla was the region primarily involved, since the patient at first presented signs of acute labio-glosso-laryngeal paralysis. The acute form of this paralysis is, indeed, most frequently caused by thrombosis of one of the vertebral arteries, or else of some one or more of the median branches of the anterior spinal (see p. 367), such an event leading subsequently to softening in the territory of the occluded vessel.

Nothing special requires to be said concerning the various Causes of Chronic Bulbar Disease beyond what has been already stated (pp. 357-362).

It only remains, therefore, to put into brief tabular form the principal indications by which we are guided to a pathological diagnosis in cases of bulbar paralysis.

ACUTE BULBAR DISEASE.	**Traumatism.**	Previous blow or fall; condition in which patient is found. Possible evidence of disease of cervical vertebræ.
	Hæmorrhage.	Abrupt onset during muscular strain, emotion, etc., in person over fifty years of age. Perhaps hypertrophy of left ventricle, pulse of high tension, and evidence of renal disease.
	Embolism.	Abrupt onset in child or young adult, the subject of valvular disease. Symptoms unilateral, and most likely referable to left side of bulb.
	Thrombosis.	Not abrupt, or symptoms increasing for a short time. If in young person, during or after some acute disease; if in person over 50, probably with evidence of arterial degeneration, and weak, irregularly-acting heart.
CHRONIC BULBAR DISEASE.	**Chronic Meningitis.**	Evidence of involvement of two or three nerve roots of bulb. Previous history of syphilis probable.
	Tumour.	Existence of general signs of tumour, viz., cephalalgia, sickness and optic neuritis; plus various signs of bulbar disease.
	Disseminated Sclerosis.	Absence of general signs of tumour. Evidence of some dissecting lesion. Common coincidence of spinal, bulbar, and other encephalic symptoms.
	Labio-Glosso-Laryngeal. Paralysis.	The combination of symptoms indicative of this disease, of progressive type, in a person over 40 years of age, who may or may not also be the subject of progressive muscular atrophy.

REGIONAL DIAGNOSIS.

Diagnostic Indications Derivable from a Consideration of the Blood Supply of the Bulb.

A reference to Figs. 70-72 and to the account given of the blood supply of the bulb, will show the extent to which it is dependent upon the vertebrals, and the great importance of the median arteries given off from the principal branches of the vertebrals, viz., the anterior spinal arteries. It will show, also, that some of the median arteries—*e.g.*, those furnished to the upper part of the medulla—are given off from the first part of the basilar artery. These vessels, known as the sub-protuberantial arteries (Fig. 71, 2,2′,2″,) supply the important nuclei of the vagus and of the glosso-pharyngeal nerves.

For a more detailed account of embolism and thrombosis in the vessels of the bulb, together with references to many cases, see Duret in 'Archiv. de Physiologie,' 1873, p. 109.

(a). When a thrombosis exists in one or other of the **vertebral arteries**, so situated as to cut off the blood supply from its anterior spinal branch, this entails a similar cutting off of blood from the corresponding median branches, which supply the nuclei of origin of the spinal accessory and of the hypoglossal as well as the lower nucleus of the facial. The consequence is that such a pathological accident will give rise to the symptoms of a labio-glosso-laryngeal paralysis of acute origin. At the same time, there may be more or less paresis of the limbs on the side affected. In such a case as this, although the paralysis of the mouth, of the tongue, as well as of the arch of the palate and of the larynx would be unilateral, still it has been found that complete aphonia and dysphagia have been produced.

But, owing to certain not uncommon vascular anomalies, it may happen that thrombosis of one vertebral leads to a bilateral paralysis of the three nerve nuclei above mentioned. This is most likely to occur when thrombosis or embolism, but especially the former, involves the left vertebral, because it not unfrequently happens that only one anterior spinal artery exists, which is then commonly given off from the left vertebral artery. In such a case, the thrombosis of this vessel would give rise to an acute, typically complete, labio-glosso-laryngeal paralysis, in association with some amount of paresis of all four limbs.

Even a small hæmorrhage from one of the median arteries of the bulb that supplies blood to the nuclei of the spinal accessory or the hypoglossal, has also been known to cause most of the symptoms of a labio-glosso-laryngeal paralysis of abrupt onset. At the autopsy on a case of this sort recorded by Cornillon, the sole lesion found was a small extravasation of blood, about equal in volume to a pea but having a triangular shape. It was situated in the median line its base being close upon the floor of the fourth ventricle, whilst its apex was near the antero-inferior aspect of the bulb. Small foci of softening have also been found, in other cases, having much the same size and mode of distribution.

(b). Where the **first part of the basilar artery** becomes obstructed either by thrombosis or by embolism, the result is a cutting off of the blood supply from the sub-protuberantial arteries which nourish the all important nuclei of the pneumo-gastric nerves. If this cutting off of the blood supply be abrupt and complete, or should a minute hæmorrhage take place into the substance of one of the vagal nuclei, from rupture of one of the sub-protuberantial arteries—in either case the result may be a sudden or at least a rapid death. It is especially where this portion of the basilar artery becomes thrombosed that rapid death is known to occur. In addition to the cases cited by Hayem ('Archiv. de Physiologie,' 1868), the author has recently recorded another remarkable example of this class (' Trans. of Clin. Soc., 1885). In these cases, there is paralysis of both sides of the body, face included, with general flaccidity of muscles throughout the whole body; abolition of all reflexes in the lower extremities; face, pale, cold and clammy (possibly cyanosed); respirations often greatly embarrassed and irregular (of Cheyne-Stokes type), or else extremely rapid (even as high as 105 per minute, with a pulse rate of 150). The rectal temperature soon after the onset may sink as low as 94° or 95° F.

Signs and Symptoms of Lesions in the Bulb.

Owing to the exceedingly complex structural and physiological relations of the bulb, the symptomatology due to lesions in it, is altogether unusually subject to variation in different cases. In dealing with such a subject the most useful course to pursue, will be to indicate some of the most common combinations of symptoms, upon the existence of which we should be warranted in arriving at the diagnosis that a lesion exists in the bulb; mentioning, also, some of the rarer symptoms that may at times be met with.

The reproduction here of certain diagrams and figures illustrating the structure of the bulb, will serve two purposes. First, it will enable the student or practitioner more clearly to comprehend than he might

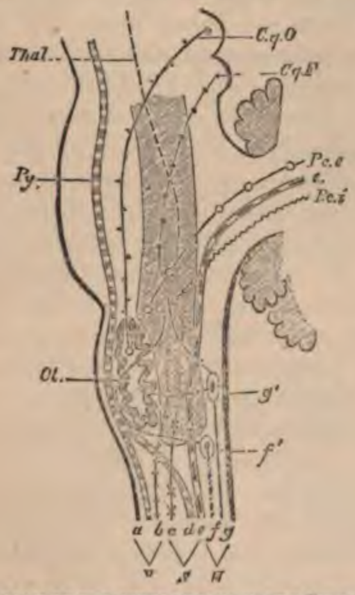

FIG. 82. DIAGRAM OF THE CHIEF TRACTS IN THE BULB [after Erb].

The formatio reticularis is represented by shading.
Ol, Olivary body. V, Anterior S, lateral, and H, posterior spinal funiculi.
Py, Pyramidal tract, dividing below into *a*, pyramido-anterior and *d*, pyramido-lateral tracts. *b*, Remainder of anterior column. *c*, Remainder of lateral column.
e-e, Direct (lateral) cerebellar tract.
f, Funiculis gracilis; and *f'*, nucleus of the same.
g, Funiculis cuneatus; and *g'*, nucleus of the same.
P.c.i., Internal fasciculus of the lower cerebellar peduncle.
P.c.e., External fasciculus of the same.
C.q.F., Tract from corpora quadrigemina to formatio reticularis.
C.q.O., Tract from olivary body to corpora quadrigemina.
Thal. Tract to the thalamus opticus.

otherwise do, the reason of the combinations referred to; and secondly, a reference to them, as well as to Figs. 73 and 103, may assist in the interpretation of other unspecified combinations of symptoms (which may be extremely varied), due to small lesions in this or that part of the bulb—either superficial or within its substance.

Labio-glosso-laryngeal paralysis is not only one of the commonest but also one of the most distinctive of bulbar affections. A brief *résumé* of its clinical characteristics, may well, therefore, take the first place as an indication of typical symptoms pointing to disease of the bulb.

The earliest signs are commonly dependent upon a commencing paralysis of the tongue. Articulation becomes indistinct, especially owing to the imperfect utterance of dental and palatine sounds in the production of which the tongue is largely concerned. Subsequently the lips become weak, and speech grows still further indistinct, owing to the defective utterance of such vowel sounds as *o* and *oo*, as well as the labial explosives *p* and *b*. After a time, difficulty in articulation becomes aggravated by paresis of the palate, with the result of giving a too uniform nasal intonation to the already very indistinct utterance. Meanwhile, the power of moving the tongue has become less and less, and some amount of atrophy of its substance may have declared itself. Deglutition gradually becomes more and more impaired, first from the increasing paralysis of the tongue,[*] but also from the paresis which supervenes in the muscles of the palate and pharynx. In later stages, the soft palate hangs motionless, so that, during the act of swallowing, fluids are apt to regurgitate into the nasal cavities. Owing to paresis in the lower facial region, the face wears a blank expressionless aspect, the lips are partially separated, there is a frequent overflow of saliva, and the patient is quite unable to whistle. When the laryngeal muscles become paralysed, the voice becomes weaker and weaker; the glottis cannot be closed, and the act of coughing becomes more and more abortive, although attempts of this sort may frequently be excited by fluids or food particles getting into the larynx. When defective phonation is at last added to extreme difficulties in articulation, speech becomes well nigh unintelligible. Comparatively early in this disease, another sign may be detected when sought for which is of some diagnostic importance—that is, the absence of all reflex contraction when the palate or the

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[*] The associated movements of the tongue during deglutition may still be preserved to some extent, when power of moving the organ voluntarily has been lost. According to Duval's hypotheses, the latter movements, and those concerned in speech generally, are governed by the nucleus of the hypoglossal itself, while the associated movements of the organ excited during deglutition, are governed rather by the accessory nucleus of the hypoglossal (Fig. 84, N'H'). (Grasset 1881, p. 513.)

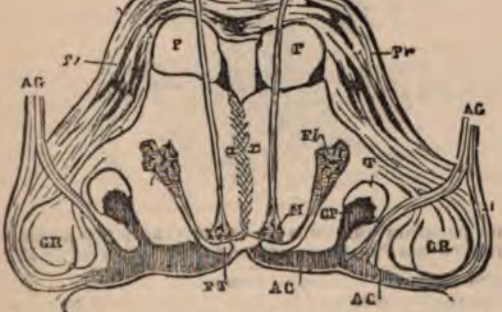

FIG. 83. DIAGRAM OF A SECTION AT THE LINE OF JUNCTION BETWEEN THE BULB AND THE PONS [after M. Duval].

P,P, Anterior pyramids. Pr,Pr, Transverse fibres of the pons between the layers of which masses of grey matter are irregularly disposed.

ME,ME, Roots of the external recti (6th nerves). M, nucleus of the sixth nerve, with which the root fibres of the facial nerve come into close relation (see Fig. 80, xvii).

FT, fasciculus teres (portion of the loop of the facial nerve).

Fi, Inferior nucleus of the facial (in which the fasciculus teres takes its origin).

GP, Head of the posterior cornu. T, Ascending root of the trigeminus.

AC, Grey substance of the floor of the 4th ventricle (nucleus of the auditory).

AG, Root fibres of the auditory nerve.

GR,GR, Restif orm bodies.

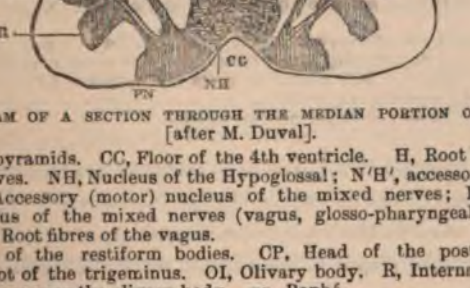

FIG. 84. DIAGRAM OF A SECTION THROUGH THE MEDIAN PORTION OF THE BULB [after M. Duval].

P,P, Anterior pyramids. CC, Floor of the 4th ventricle. H, Root fibres of the hypoglossal nerves. NH, Nucleus of the Hypoglossal; N′H′, accessory nucleus of the same. S, Accessory (motor) nucleus of the mixed nerves; PN, principal (sensory) nucleus of the mixed nerves (vagus, glosso-pharyngeal, and spinal accessory). M, Root fibres of the vagus.

NR, Nucleus of the restiform bodies. CP, Head of the posterior cornu. T, Ascending root of the trigeminus. OI, Olivary body. R, Internal, and T, external grey nucleus near the olivary body. *xx*, Raphé.

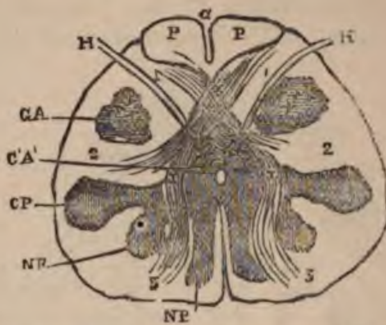

FIG. 85. SECTION THROUGH THE BULB AT THE LEVEL OF THE UPPER PART OF THE DECUSSATION OF THE PYRAMIDS—SENSORY DECUSSATION [after M. Duval].

a and *p*, anterior and posterior median fissures.

CA, Head of the anterior cornu; C'A', base of the anterior cornu (nucleus of the hypoglossal nerve); H, root fibres of the hypoglossal. CP, Posterior cornu. NR, Nucleus of the restiform body. PP', Anterior pyramids. NP, Nucleus of the posterior pyramid.

1,2,3, Antero-internal, lateral, and posterior columns of the Bulb. *x*,*x*, Fibres issuing from the posterior columns and decussating in ¹*x*.

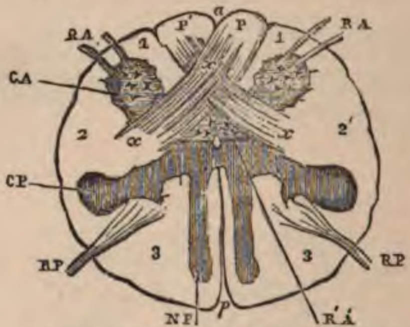

FIG. 86. SECTION THROUGH THE LOWER PART OF THE BULB AT THE LEVEL OF THE DECUSSATION OF THE PYRAMIDS—MOTOR DECUSSATION [after M. Duval].

1,2,3, Antero-internal, antero-lateral, and posterior columns. P,P', Anterior pyramids decussating at *x*, and each passing into the lateral column of the opposite side.

RA, Anterior roots of nerves. CA, Head of the anterior cornu, which is separated by the decussating fibres from R'A', the more central portion of the anterior grey matter. RP, Posterior roots of nerves. CP, Posterior cornu. NP, Nucleus of the posterior pyramids.

a and *p*, Anterior and posterior median fissures.

back of the pharynx is touched with a feather or other suitable object; at a later stage of the disease, a similar loss of reflex excitability may be found to exist in the vocal cords themselves.

In the terminal stages of the disease we may have signs of implication of other nerve nuclei. But these collateral or late results, are of comparatively little importance in regard to diagnosis, although respiratory and cardiac troubles which are to be enumerated among them, constitute all-important modes by which the lives of such patients are ultimately brought to a close.

Extreme difficulty or loss of the power of articulating together with some difficulty in swallowing, with or without some amount of paresis (bilateral) of limbs, may be occasioned by symmetrical bilateral lesions in the cortex of the cerebral hemispheres, occupying the posterior extremity of the third frontal and the inferior extremity of the ascending frontal convolutions (Fig. 54); or, also, by the double implication of the efferent fibres from these convolutions when on their way towards the genu of the internal capsule (Lépine). Such cases may be distinguished by the fact that there is a history of two attacks; by the fact that mere phonation is not affected; and also by the fact that the reflex movements of the palate and pharynx, when touched by some foreign body, are found not to be interfered with. Respiration and the action of the heart are, also, not interfered with to the extent that they would be where we have to do with morbid processes in the bulb.

Apart from the special form of bulbar disease above referred to, we may also look upon the following combination of signs as strongly indicative of a lesion in the bulb, though a somewhat similar grouping of signs is apt to be met with in cases of disease in the immediately contiguous brain territory, the pons. The mode of distinguishing between lesions in these two sites will be subsequently dealt with. The signs referred to are these :—More or less marked difficulties in articulation and deglutition associated with diminished movements of the tongue, and marked impairment of reflex contraction on touching the palate or back of the pharynx ; impaired phonation and diminished power of coughing ; an undue tendency to laugh or cry on slight provocation ; an unnaturally slow and irregular or a too frequent action of the heart ; unnaturally slow, frequent, or irregular respirations (possibly of the Cheyne-Stokes type) ; the possible coexistence of diabetes, polyuria, or albuminuria ; no distinct anæsthesia, but possibly hyperæsthesia or paræsthesiæ in different parts of the body ; a blank expressionless face ; more or less marked bilateral paresis of limbs together with partial paralysis of the trunk muscles—though in other cases the paralysis may be unilateral, or it may principally affect both lower extremities ; the occasional existence of early rigidity in the paralysed limbs, with notable exaggeration of the so-called deep reflexes ; convulsions or vomiting are occasional symptoms; mental power is not appreciably impaired, although it may seem to be by reason of the marked diminution of emotional control above referred to; and, finally, diminished control over the sphincters of the bladder and rectum, where the paralysis is bilateral.

The symptoms enumerated above are those which are best known to occur in association with bulbar affections of different kinds. It is scarcely to be expected that all would occur in the same case; but that different groups of them would be met with in different cases, the precise combination of which would vary in accordance with the exact situation and extent of the lesions existing in this or that case. Although the bulb is known to contain the principal vaso-motor centres, yet clinical medicine has not hitherto thrown any definite light upon the effects produced by their damage or irritation. The bulb is also in all probability traversed by two sets of fibres emanating from supposed 'heat-regulating centres' situated in the parietal region of each hemisphere (see p. 158); these same fibres must likewise traverse the pons. It might be supposed, therefore, that marked depressions or marked elevations of temperature might be equally prone to occur from lesions in the pons and in the bulb; yet it is not so. Such elevations and depressions of temperature are much more frequently observed in association with diseases of the pons than with those of the bulb. Perhaps this seeming anomaly may be in great part explained by the greater absolute frequency of disease in the pons, especially of those morbid changes which are characterized by an abrupt onset (hæmorrhages and occlusions of vessels), and which are at the same time of large extent—these being the conditions most likely to lead to such notable depressions or elevations of temperature. Nevertheless, in some cases of bulbar disease an extreme terminal rise of temperature has been noted (Leyden, Erb). I have already (p. 160) advanced some reasons for believing that no decussation of the vaso-motor channels, or of the fibres which influence heat production throughout the body, takes place in the bulb or elsewhere; but much light is capable of being thrown upon this question by subsequent careful clinical examination of patients suffering from unilateral lesions in the bulb, pons, and cerebral peduncles.

It should be borne in mind, that in some cases of unilateral lesions of the bulb, we may have to do with paralyses of some one or more of the bulbar nerves, but especially of the hypoglossal, on the side of lesion, together with paresis or paralysis of limbs (not of the face) on the opposite side owing to an implication of the anterior pyramidal fibres above their point of decussation.

It has been already mentioned, that there may be a great general similarity between the symptoms occasioned by disease in the pons and disease in the bulb. Certain symptoms may be common to both, such as difficulties of a marked character in articulation and deglutition associated with bilateral paralysis more or less complete in degree, together with loss of emotional control; on the other hand, there are often distinguishing signs pointing to the existence of a lesion in one locality rather than in the other. Some of the principal differential signs are these:—

Combination of Signs pointing to a Lesion in the Pons.

The co-existence of marked difficulty in deglutition and in articulation, with an extremely well-marked paralysis of the facial muscles, either on the opposite or on the same side as the paralysis of limbs.

No loss of phonation or paralysis of the palate; preservation of its reflex, and of that of the pharynx.

Evidence of paralysis of the fifth or of the sixth nerves. The existence of well-marked sensory defects in the paralysed limbs or half of the body.

Strabismus, external or internal, from paralysis or irritation of the sixth nerve.

Combination of Signs pointing to a Lesion in the Bulb.

The coexistence of extreme difficulty in articulation and in deglutition, with paresis involving the lower facial muscles only (especially the orbicularis oris), and generally of a bilateral type.

Often, loss or great impairment of phonation, with paralysis of the palate, together with loss of its reflex excitability and also of that of the pharynx.

Evidence of defects of hearing of centric type.

Absence of well-marked sensory defects in the paralysed limbs.

Marked interference with the action of the heart, or disturbance of the respiratory rhythm.

PART III.

PARALYSES DUE TO LESIONS OF THE CRANIAL NERVES.

PRELIMINARY REMARKS.

Under this general heading we propose to consider in detail the facts of importance concerning paralyses due to lesions of each of the different cranial nerves, whether the lesion affects the extra- or the intra-cerebral course of the several nerve fibres.

The recognition of the signs of paralysis of this or that cranial nerve, or of this or that group of such nerves, where such signs are present, is always a matter of great importance, with a view to the correct diagnosis of the localisation and even of the nature of organic diseases of the brain.

It is impossible, however, that this important subject could be adequately treated in the sections dealing with the problems of Pathological and Regional Diagnosis as applied to Encephalic Diseases generally. The attempt to do so would, moreover, have involved much needless repetition.

For these reasons the whole subject is dealt with here as a separate part of this work, in order that reference may be made to it when any detailed information is needed—either concerning the signs of paralysis of any given cranial nerve, or concerning the common conditions under which such paralyses are apt to be met with.

An independent section is devoted to each cranial nerve, with the one exception of the motor nerves of the eyeball, which, on account of their close functional relations, are dealt with together in a single section.

These paralyses due to lesions of different cranial nerves are considered in the following order :—

A.—Olfactory Nerves.
B.—Optic Nerves.

C.—THIRD NERVES.
FOURTH NERVES.
SIXTH NERVES.
D.—FIFTH NERVES.
E.—FACIAL NERVES.
F.—AUDITORY NERVES.
G.—GLOSSO-PHARYNGEAL NERVES.
H.—PNEUMO-GASTRIC NERVES.
I.—SPINAL ACCESSORY NERVES.
J.—HYPOGLOSSAL NERVES.

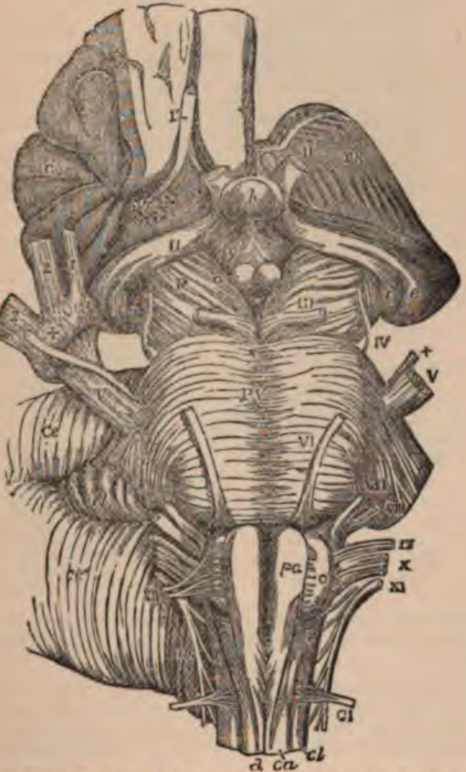

FIG. 87. PARTS ABOUT BASE OF BRAIN SHOWING RELATIONS OF CRANIAL NERVES [after Allen Thomson]. Natural size.

On the right side the convolutions of the Island of Reil have been left, together with a small part of the anterior cerebral convolutions. On the left side these have been removed by an incision carried between the Thalamus and the Cerebral Hemisphere.

1′, The Olfactory tract cut short and lying in its groove. II, Left Optic Nerve in front of the commissure. II, The right optic tract. Th, The cut surface of the Left Thalamus. C′, Island of Reil. Sy, Fissure of Sylvius. *xx*, Anterior perforated space. *e* and *i*, The external and internal corpus geniculatum, respectively.

h, Pituitary body. *tc*, Tuber cinereum with the Infundibulum. *a*, One of Corpora albicantia. P, Crus cerebri. *f*, The fillet. III, Third nerve. *x*, The posterior perforated space.

PV, Pons Varolii. V, The greater root of the fifth nerve; *x*, its lesser or motor root. VI, Sixth nerve. VII, The facial. VIII, The auditory. IX, The glosso-pharyngeal. X, the pneumo-gastric. XI, The spinal accessory nerve. XII, The hypoglossal.

CI, The suboccipital or first cervical nerve. *pa*, Pyramid. *o*, Olive. *d*, Anterior median fissure of the cord, above which the decussation of the pyramids is represented. *ca*, Anterior column of cord. *r*, Lateral tract of bulb continuous with *cl*, the lateral column of the spinal cord.

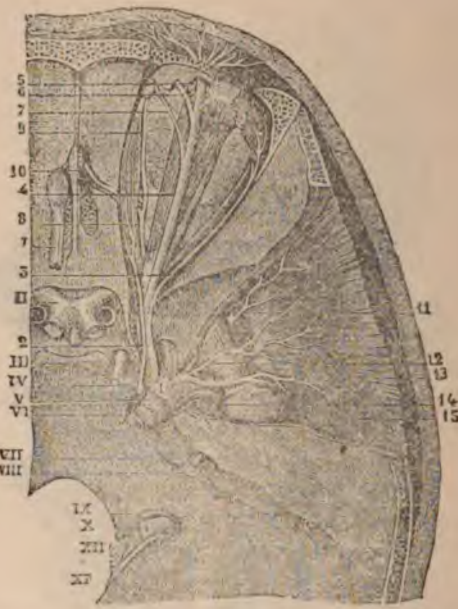

FIG. 88. VIEW FROM ABOVE OF THE NERVES OF THE ORBIT AND OTHER CRANIAL NERVES NEAR THEIR POINTS OF EXIT FROM THE SKULL [after Hirschfeld and Leveillé].

I, Olfactory tract passing forwards into the bulb. II, Optic Commissure. III, Oculo-motor. IV, Trochlear Nerve, V, Large root of the fifth nerve; a small portion of the lesser root is seen below it. VI, Sixth nerve. VII, Facial. VIII, Auditory. IX, Glosso-pharyngeal. X, Pneumo-gastric. XI, Spinal accessory. XII, Hypoglossal.

1, Gasserian ganglion; 2, ophthalmic nerve; 3, lachrymal nerve; 4, frontal; 5, external, and 6, internal branch of the supra-orbital nerve; 7, supra-trochlear nerve; 8, nasal nerve; 9, its infra-trochlear branch; 10, nasal nerve passing through the internal orbital canal; 11, anterior deep temporal proceeding from the buccal nerve; 12, middle deep temporal; 13, posterior deep temporal arising from the masseteric; 14, origin of the auriculo temporal; 15, great superficial petrosal nerve.

A.—OLFACTORY NERVES.

In functional disorders of the brain, especially in hysteria, in epilepsy, and in insanity, various exaltations or perversions of the sense of smell are apt to be met with. In hysteria exaltations of smell are by no means uncommon. In epilepsy temporary subjective sensations of smell, and in insanity abiding hallucinations of smell, occur not unfrequently. Such conditions are, however, very rarely associated with paralysing lesions of the brain. In these latter cases, if the sense of smell is affected at all, we have mostly to do with diminutions of this sense, or with its abolition, on one or on both sides of the body (Olfactory Anæsthesia, or Anosmia).

The student must not make the mistake of testing the degree of integrity of this sense with irritating substances such as ammonia or acetic acid, which may be appreciated through the medium of the fifth nerve, but must apply first to one nostril and then to the other some less pungent odorous substance such as camphor, oil of cloves, peppermint or of lavender, alternately with something having a less agreeable odour such as tincture of valerian, sumbul, or assafœtida. It must be recollected, moreover, that the full appreciation of flavours also occurs through the medium of the olfactory nerve. The patient must be questioned, therefore, in reference to his ability to appreciate flavours. This, indeed, is the side of the defect commonly the most obvious to him, so that a patient whose sense of smell is defective may be aware of a diminished power of appreciating flavours when he is unconscious of the existence of any loss of smell.

Where, after proper trials, it is found that more or less anosmia exists on one or both sides, a certain reserve must at first be maintained in regard to the significance of this defect, owing to the following considerations :—

(1) In rare cases the sense of smell is congenitally defective. (2) In elderly persons it sometimes becomes so by reason of atrophic degenerations occurring in the olfactory fibrils. In this, as well as in the former case, the defect is likely to be bilateral, and greatly to diminish the power of appreciating flavours. (3) Any condition of the nostrils which prevents the free access of air to the upper regions of the nasal cavity may cause loss of smell, without necessarily interfering with the appreciation of flavours, because the physical causes of these latter impressions may still make their way from the back of the mouth and pharynx through the posterior openings of the olfactory chambers,

even when obstructions, due to nasal polypi, for instance, may interfere with the act of smelling.

Again it is important to remember that some diminution of the sense of smell is apt to occur as a consequence of disease of the fifth as well as of the seventh cranial nerves.

Paralysing lesions of the fifth nerve affect the sense of smell, owing to the fact that its superior maxillary division supplies the mucous membrane of the nasal cavity, and when this nerve is paralysed the mucous secreted becomes greatly diminished in quantity and viscid in character over the olfactory mucous membrane, with the effect of notably diminishing the sense of smell. This defect is most likely to occur when the fifth nerve is implicated in some part of its course beyond the Gasserian ganglion. An affection of the intra-cerebral roots of the fifth nerve would probably not at all interfere with the sense of smell (see p. 384).

In peripheral paralysis of the facial nerve, diminution, rarely loss, of smell may be entailed on the affected side, in consequence of the patient's inability to draw sufficient air into the nostril and to direct it towards the olfactory region. This cause may be strongly reinforced by another, viz., the dryness of the mucous membrane of the nostril, occasioned by the fact of the paralysis of the orbicularis palpebrarum permitting an overflow of tears, rather than their passage into the corresponding nasal cavity. It would seem that any diminution that occurs in what we may call the passive exercise of the sense of smell (as, for instance, in the recognition of volatile substances) in cases of facial paralysis, must be due to this latter cause.

Pathological Diagnosis.

The sense of smell may be impaired by blows and falls upon the head. Such traumatisms may tear the delicate olfactory nerves away from the bulb, with or without the occurrence of fracture of the frontal and ethmoid bones, or they may possibly cause bruising of the tips of the temporo-sphenoidal lobes (see p. 264).

The sense of smell may also be impaired or destroyed, on one side or on both, by disease involving the bulbs and olfactory tracts, mostly by pressure or by extension of lesions from contiguous parts. In cases of thrombosis or embolism of the anterior cerebral artery the olfactory bulb and tract may, in common with other parts within this vascular territory, undergo a process of ischæmic softening.

Again, as Prevost has shown, an independent degeneration followed by atrophy may sometimes involve the olfactory bulbs.

The morbid processes apt to involve the olfactory bulbs and tracts secondarily, are tumours originating in the brain or the meninges near them, growths extending inwards from the orbital cavity, or exostoses springing from the orbital plates. They may also be implicated by a contiguous meningitis, with or without the association of caries. Again, a large abscess or new growth, situated in the frontal or anterior part of the parietal region, may cause so much downward swelling of the brain substance and increase of pressure, as to make the anterior margin of the sella turcica deeply indent the posterior and internal parts of the orbital lobes, the indentation extending completely across the hinder extremities of the olfactory tracts, in such a way as necessarily to obliterate all functional activity. Some years ago I saw a remarkable instance of this kind of damage occasioned by a large tumour in the anterior part of the right cerebral hemisphere.

In the case of a tumour growing from the eyeball or any part of the orbital cavity, the question often arises whether the brain has become involved or not. Involvement of the part of the frontal lobe over the orbital plate, is apt not to reveal itself by definite signs; in such cases, therefore, the sense of smell on the same side should always be tested, as its loss or decided impairment would tend strongly to show that the brain in this direction had become, or was about to become, affected.

Again, in cases of thrombosis of the carotid artery, either on the right or on the left side, if the thrombus extends into the middle cerebral it will, in all probability, block the anterior cerebral also, and with it the ophthalmic artery. In such cases we may have loss of smell as well as loss of sight on the side of lesion, occurring simultaneously with a hemiplegia of the opposite side of the body.

In **cerebral hemianæsthesia**, whether of organic or of functional origin (the latter being the so-called hysterical hemianæsthesia), it is commonly found that the sense of smell is lost or impaired, like other sense endowments. Where this condition is of organic origin we now know that it is dependent upon lesions occupying the posterior third of the hinder segment of the internal capsule (p. 152). This being so, it is very difficult, with our present knowledge, to understand how fibres from the opposite olfactory nerves should be met with in this situation. Of course we may imagine with Charcot, as a mere hypothesis, that, mixed with other sensory fibres, there are in this posterior region of the internal capsule "fibres proceeding from the olfactory tract through the medium of the anterior commissure, the extremities of which, as we know from the descriptions of Burdach and Gratiolet, are directed backwards into the substance of the occipital and sphenoidal lobes" ('Localisation,' p. 118). Still, of the existence of such olfactory fibres, as Charcot admits, we have no anatomical proof at present.

The question as to the real intra-cerebral course of the olfactory nerves on their way to the cerebral cortex is a very puzzling one. Arguing from analogy, it might be expected that the olfactory fibres would decussate somewhere after penetrating the brain, so that the fibres from the olfactory bulb of one side would be brought, either wholly or in part, into structural and functional relations with the opposite cerebral hemisphere, after the manner which obtains for other sensory nerves.

There are good reasons for believing that the anterior commissure of the brain has important functions of some kind in connection with the olfactory sense. It is well known, for instance, that in the lower animals the size of the anterior commissure is in direct relation with the size of the olfactory lobes. On this subject I have elsewhere said :—"It is larger in Marsupials and Monotremes than in any other Mammals, and in higher representatives of the class it is usually thickest in those animals which have well developed olfactory lobes, since it seems to be a commissure serving principally to bring the two cerebral centres of the sense of smell into relation with one another. In part it connects the Olfactory Peduncles with one another, and in part it serves to bring into relation those regions of the brain in each hemisphere in and about the Hippocampi, to which the majority of the root fibres of such tracts proceed." . . . "The anterior commissure is a distinct band of white fibres which crosses the anterior part of the third ventricle, and on each side penetrates the substance of the Corpus Striatum. It is not, however, as it seems to be a commissure connecting these bodies. Careful dissection suffices to show that its fibres merely pass through the corpus striatum on each side (where they lie in a distinct groove or canal), that they emerge on the under and outer surface of these bodies, and that they are thence distributed to the convolutions forming the tip and inner or under surface of the temporal lobe. It is, as Broadbent says, and as other anatomists had previously recognized, a sort of accessory corpus callosum, connecting those parts of the two temporal lobes which could not otherwise be easily brought into relation with one another" ('Brain as an Organ of Mind,' pp. 271 and 454).

It should, however, be mentioned here that according to some anatomists the anterior commissure contains, in addition to the commissural fibres above referred to, the whole of the fibres of the middle root of the olfactory tract, which root-fibres decussate in the commissure—those from the right middle root emerging beneath the left corpus striatum, and *vice versâ* (Fig. 89, Coa′).

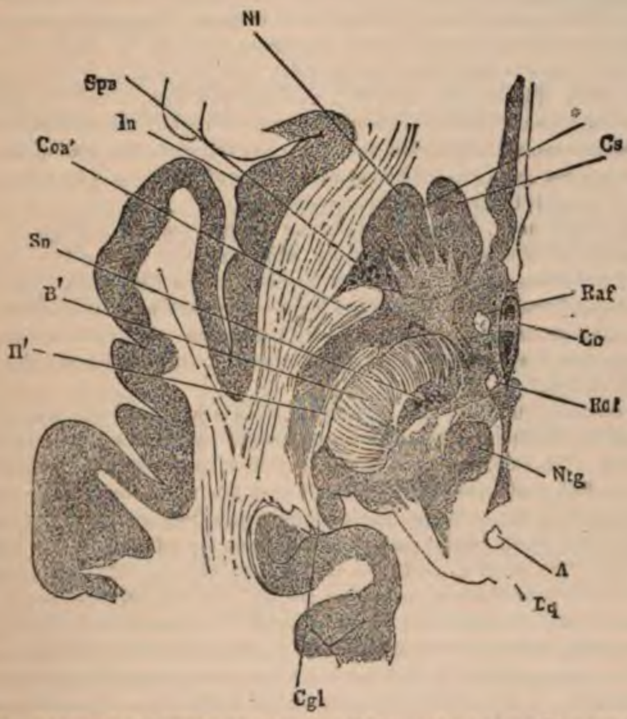

FIG. 89. HORIZONTAL SECTION OF THE LEFT HEMISPHERE OF THE BRAIN CLOSE TO ITS INFERIOR SURFACE [after Henle].

Lq, Lamina quadrigemina. A, Aqueduct of Sylvius. Ntg, Red nucleus of the Tegmentum.

Rdf, and Raf, Descending and ascending roots of the Fornix. Co, Optic commissure, seen through the floor of the third ventricle. Cs, Caudate nucleus of the corpus striatum. Nl, The lenticular nucleus. *, Division between the two nuclei of the corpus striatum. Spa, Anterior perforated space. In, Island of Reil.

Coa′, Anterior commissure. Sn, Substantia nigra. B′, Transverse section of the Crusta. II′, Optic tracts. Cgl, External geniculate body.

It is decidedly improbable, however, that all the fibres of the olfactory tracts undergo any such decussation. Many of the fibres of the outer root, in some of the lower animals especially, pass directly into the anterior extremity of the temporal lobe, and some of the fibres of this root seem to pursue the same course in man. Other fibres of the outer root of the olfactory tract are said by Alexander Hill to pass along the hippocampal region into the posterior pillar of the fornix, and thence on to the anterior pillar of the fornix, to terminate with it in a nerve nucleus (olfactory) situated in the anterior and inner part of the thalamus on the same side. How this nucleus is brought into relation with the cerebral cortex is altogether uncertain.

It is well known that Ferrier regards the convolutions about the tip of the temporal lobe (subiculum) as the cortical terminus for olfactory impressions. If we assume that a decussation of olfactory fibres takes place in the anterior commissure, then such fibres in order to reach the tip of the temporal lobe would have to come round not very far from the hinder or outer border of the crusta (Fig. 89, Coa′ and B′), and thus would not be very far removed from other afferent fibres in the crusta or posterior part of the internal capsule. This possibly explains why, when lesions involve this particular region, we may, as we often do, get a crossed impairment or loss of smell, as well as of other modes of sensibility (cerebral hemianæsthesia).

On the other hand, according to Ferrier ('Functions of the Brain,' p. 185), destruction of the subiculum (or more precisely of the tip of the uncinate convolution), in the monkey, causes diminution or abolition of the sense of smell *on the side of the lesion*—thus confirming, as he thinks, the direct relations of this sensory nerve with the cortex. He believes "there is no anatomical basis of cross connection between the olfactory bulbs and their cerebral centres."

It is certainly quite true that the sense of smell is just that mode of sensibility in which no discrimination is ever made between impressions coming from the two sides. No embarrassment would, therefore, seem likely to be produced from the fact that impressions of smell from the right nostril are brought into relation in the corresponding cerebral hemisphere with gustatory, visual, auditory, and tactile impressions emanating from the left side of the body, or *vice versâ* ('The Brain as an Organ of Mind,' pp. 536 and 482). This view of Ferrier's, however, seems at present to conflict with clinical experience; since the loss of smell in some cases of cerebral hemianæsthesia is more than can be accounted for solely by loss of functional activity in the fifth nerve, which, as already stated (p. 380) would lead to some diminution of this mode of sensibility, if the paralysing lesion involved the roots of the fifth nerve beyond the Gasserian ganglion. Such a defect is, however, less likely to arise if the intra-cerebral roots of the fifth nerve only are involved (as would be the case when lesions or functional changes induce cerebral hemianæsthesia). Ferrier's attempted explanation of the discrepancy can therefore scarcely be deemed adequate.

B.—OPTIC NERVES.

What we have to say here concerning the optic nerve must be supplementary to what has already been said in speaking of some of the signs of brain disease (pp. 134-150).

We there discussed, principally, the conditions under which Hemiopias of different kinds and Crossed Amblyopia showed themselves, as results of intra-cranial or of cerebral disease in particular; and we more briefly alluded to the conditions with which optic neuritis and optic atrophy are associated.

We now propose to speak principally concerning Amblyopia and Amaurosis and the symptoms by which they are characterized and, subsequently, concerning Optic Neuritis and Optic Atrophy, with especial reference to their characteristics and modes of causation.

The result of *irritation* of the optic nerve fibres or of their intracerebral roots or centres are of slight importance, as compared with that of lesions which cause a diminution of functional activity in these same fibres or centres. Still, it must not be forgotten that, many effects belonging to this former category are met with from time to time, varying from illusions or hallucinations of the sense of sight (as a result of unnatural action of the cerebral centres), down to mere flashes of light or the production of luminous discs or rings, caused by irritation of the optic nerve fibres themselves.

Where there is *diminished functional activity* of the optic nerves or centres, leading to diminution of sight, the condition is known as *Amblyopia;* while when sight is lost from the same cause, the condition is named *Amaurosis.*

Amblyopia.—Whether we have to do with a functional amblyopia (such, for instance, as may be associated with hysterical hemianæsthesia) or with one which is caused by structural disease, the symptoms by which the condition is characterised are precisely the same. In almost all cases of amblyopia we meet with these three distinct alterations in regard to vision :—(1), Diminution in the acuity of vision ; (2), Diminution in the field of vision ; (3), Diminution in the power of perceiving differences of colour.

Something must now be said in regard to each of these components of the amblyopic state, more especially as to the simplest methods of estimating the extent to which they exist. It would be inappropriate here to attempt to enter into refinements in reference to this subject ; these may be found, when needed, in works on ophthalmic medicine ; here we shall only refer to such methods of investigation as may be adequate for those who are not specialists.

(1).—*Diminution in the Acuity of Vision.* Acuity of vision is judged in any given case by the ability of the patient to discriminate differences of form (commonly those of letters) at definite distances proportionate to their size. It is normal only when the image of the object looked at falls upon the yellow spot of the retina, when it is clearly defined, sufficiently bright, and not below a certain minimum size.

The needful principles to be known in reference to this subject, have been well and briefly put by Nettleship, in the following manner. The size of the image depends (*a*) upon the size of the 'visual angle' enclosed by the two lines drawn from the extremities of the object to the 'nodal point' just behind the crystalline lens; and (*b*) on the distance of the nodal point from the retina, which in the normal eye is 15mm. The form of any letter or character is distinguished by a properly formed and healthy eye, with average light, if it subtend a visual angle of five minutes, each of its separately distinguishable parts subtending an angle of one minute. If the nodal point be more than 15mm. from the retina, the image will be larger, and acuteness of vision therefore increased; this occurs in myopia, and also when a convex glass is held before the eye. The reverse is true if the distance be less than 15mm., as in hypermetropia, and when a concave glass is held before the eye. Vision or 'fixation' is termed direct or central when the image of the object looked at falls on the yellow spot; indirect or excentric when, in consequence of impairment of function at the yellow spot, an image falling on some other part is better seen. The clearness of the image depends (opacities of the media apart) upon the retina being exactly at the focus of the refracting media of the eye; it is also influenced somewhat by the size of the pupil, being, other things equal, better when the pupil is small.

Acuteness of vision is commonly tested by requesting the patient to read type of a certain size at a certain given distance. For this purpose test-types are in common use, composed of letters and words of different but definite sizes, so that each subtends the minimum angle of five minutes when seen at the number of feet (or mètres) which is specified by a figure above each specimen. Where the acuity of vision is normal it is expressed as unity (V. = I); where it is subnormal it is expressed as a fraction—the denominator of which is the distance in feet (or mètres) at which the particular type with which trial is being made ought to be seen by a normal eye, whilst the numerator corresponds with the number of feet (or mètres) at which it can be seen by the patient in question. Thus, suppose we are examining the acuteness of vision of a patient with a type which should be read by a normal eye at 20 feet, and we find that with the right eye he cannot read the type at a greater distance than 18 feet, and with the left eye not at a greater distance than 10 feet, then in our notes we should express these facts as follows :—$V = \frac{18}{20}$ R, and $\frac{10}{20}$ L.

The acuteness of vision is said to become progressively lower in healthy persons after the age of sixty, so that at eighty it is only about $\frac{1}{2}$ (Donders).

(2).—*Diminution of the Field of Vision.* What is called the visual field is the whole surface visible to one eye singly whilst at rest.

Nettleship says:—"It forms a concave surface, all the points of which are equidistant from, and perpendicular to, their corresponding points on the retina. In the outward and downward part it reaches to 95° from the centre; inwards, upwards, and downwards only to about 60°. Projected on a flat surface it thus forms an oval. The centre of the field ('fixation point') corresponds to the yellow spot, and the 'blind spot' is about 15° outwards from this point."

For accurate measurement of the field of vision an instrument known as a perimeter must be employed, but where one of these is not accessible we may still with comparative ease obtain important information in regard to the field of vision in one or other or in both eyes of our patient. Thus, he may be directed to close one eye and to look fixedly with the other at a white spot on a black board about one foot off; the observer then moves a piece of chalk towards different points of the periphery of an imaginary circle described round this spot, noting in each case, by a dot on the board, the situation at which the chalk becomes just visible. When an examination of this sort has been completed, a line joining the various points all round will mark out the visual field in a fairly accurate manner, where the operation has been carefully conducted and a sufficient number of peripheral points have been noted.

Another still rougher method is the following, which may be employed at times for revealing any great narrowing of the visual field in particular directions. The patient stands with his back to the light, and covering one eye, looks steadily from a distance of eighteen inches at the corresponding eye of the observer, whilst the latter moves his hands to the right the left, above, below and in diagonal directions to different distances from the fixed point towards which the patient is looking. Where the field of vision is limited in any particular direction, the operator will find that he has in this direction to bring his hand or forefinger nearer to the fixed point (to an extent which varies with the degree of narrowing of the field), in order that the hand may be distinctly seen. In this way a very distinct defect in any particular direction may be easily detected.

The most common alteration in the visual field consists in a concentric narrowing which, beginning at the periphery, may increase gradually until at last only a small central area is left.

At other times, the first loss of vision may appear in the centre of the field, constituting what is known as a central scotoma. Here a dark spot of varying form appears, and may gradually extend in area. Or a number of small scotomata may be scattered irregularly over the field of vision.

Gowers calls attention to a theory of Leber by which he seeks to account for the common modes of narrowing of the visual field. The former writes:—('Medical Ophthalmoscopy,' p. 98):—"Leber has suggested that the affection of the periphery or centre of the field is due to the position of the lesion in the optic nerve, on the theory that the peripheral fibres of the nerve end soonest on the retina, and the central fibres of the nerve which are most superficial in the papilla, go to the peripheral parts of the retina. Hence he assumes that an affection of the circumference of the nerve causes a central loss in the field of vision, and an affection of the centre of the nerve a peripheral affection of the field. At present this is little more than a theory. We cannot rely on the regularity of the arrangement of the fibres in the papilla."

Sometimes the narrowing of the field from the periphery may proceed so irregularly as to lead to a sector-like defect; at other times one or other lateral half, or an upper or lower half, of the field of vision may be obliterated. In the former of these cases we may suppose that pressure, or disease of some kind, acts upon particular segments of the nerve trunk; while in the case of lateral defects, we mostly have to do with disease or with arrested functional activity either in some part of the chiasma or of one of the crura cerebri.

(3).—*Diminution of Colour Vision.* On rare occasions, even marked amblyopia may not be associated with any defect in colour vision; as a rule, however, it is otherwise. On the other hand, it is well known that in many cases defects in colour vision may be congenital, and not related to either of the two kinds of defect of which mention has already been made.

In reference to this subject it is important to bear in mind that a great natural difference exists in the size of the field of vision for different colours. Compound colours are generally lost sooner than their constituents, when they are moved from the centre of the field towards the periphery; while, of the four simple or fundamental colours, green is usually lost first, then red, then yellow, and lastly blue, which is perceived nearly up to the edge of the field for white. Thus, if the distance at which each colour ceases to be distinguished in the different parts of the field, be marked upon the same chart we obtain a series of more or less concentric lines. The difference between the several colour fields is exaggerated when the examination is made in a dull light, and minimized under the influence of a bright light.

When, therefore, in cases of amblyopia of functional or of structural origin, there is diminution of the colour sense, it is found that the simple colour first lost in passing from the centre to the periphery of the retina is green, then red, yellow, and blue. Irregularities in the order are, however, met with from time to time. Gowers points out that violet, instead of disappearing even before green as it often does, may persist to the last and disappear only after yellow or blue; red also may disappear before green. He refers to a patient suffering from locomotor ataxy with advanced optic atrophy who stated that "the first loss of the sense of colour of which he was conscious, was that he could see no colour in a scarlet geranium. Red gravel looked grey to him. Soon after the grass also looked grey, and he could not at a little distance distinguish it from the gravel. When examined the only colour to be recognized was violet, which he said looked blue. A medium blue was seen as white."

What has been said above concerning acquired limitation of the visual colour field must not be confounded with *Colour Blindness.* This is usually a congenital condition, not related to any other kind of visual defect, which is met with much more commonly in males (3 to 5 per cent.) than in females (·2 per cent. or less). It is shown by more or less loss of power to distinguish between certain complementary colours. Red and green are colours commonly confused; the ability to discriminate between blue and yellow being rarely interfered with. Many degrees of colour blindness exist.

There are two principal means of testing the integrity or the reverse of colour vision.

(a). We may ascertain whether the patient can or cannot identify and name certain colours. Concerning this test Gowers makes the following remarks. He points out that the method has "considerable drawbacks arising from the ignorance of the names of colours which many patients present, and from the difficulty in ascertaining, especially in the case of the congenitally colour-blind, how far the terms they use correspond in meaning with the same terms as employed by those who possess normal colour-vision. In the case of *acquired colour-blindness,* however, especially when one eye only is affected, and in those who previously (as most women) possessed an accurate knowledge of the names of colours, this method possesses special advantages which have certainly been underrated by some recent writers, since it affords information to be obtained in no other way, of the character of the perverted sensation of such patients."

(b). But the mode of examination most generally useful is that which has been perfected during recent years by Holmgren of Upsala. It consists in the comparison of colours; a carefully chosen assortment of coloured wools being provided, in which there are several shades of each colour. Certain colours are selected and the patient is asked to match them successively from other wools spread out before him. The mode in which he does this, soon affords the information needed as to the degree and nature of the colour defect, if any.

Amblyopia and **Amaurosis** occur in two classes of cases which it is convenient to distinguish artificially from one another. (1). It occurs without any apparent change in the fundus of the eye detectable by the aid of the ophthalmoscope; and (2) in association with very decidedly abnormal conditions of the optic discs, as revealed by the ophthalmoscope.

(1). Although in a very large proportion of the cases in which Amblyopia and Amaurosis occurs without any apparent change in the fundus of the eye, we should, in all probability, have to do with mere temporary and functional defects, yet it is by no means so in all of them. Where crossed amblyopia occurs for instance, in connection with cerebral hemianæsthesia due to structural disease, it is well known that no appreciable changes are, as a rule, to be detected in the corresponding eye with the ophthalmoscope (p. 135). In cases of this kind, therefore, the decision whether we have to do with structural disease or with a mere functional defect as the cause, must be made upon general grounds, and cannot be based upon the results of ophthalmoscopic examination.

The classification of this first group of cases will, in fact, be facilitated by dividing them into two sub-groups, viz., those in which there is *(a) unilateral amblyopia, without ophthalmoscopic change;* and those where there is *(b) bilateral amblyopia without ophthalmoscopic change.*

(*a*). Here we have to do with crossed amblyopia associated with cerebral hemianæsthesia (of structural or of functional origin). A hemiplegia initiated by convulsions, may be followed for a time by more or less complete blindness of one eye.

Again, unilateral blindness occurring alone in paroxysms, and generally lasting only a few minutes, is sometimes observed even in the midst of what appears to be ordinary health as a kind of abortive epileptic attack.

Dr. Ross says ('Diseases of the Nervous System,' Vol. I., p. 314), "I myself suffered for upwards of ten years from this affection. The blindness was preceded for a few seconds by a feeling of tension and fulness of the right eye, then the central portion of the visual field became suddenly clouded, but the darkness spread with such rapidity that the right eye appeared to have become almost instantaneously blind. This affection lasted from one to three minutes. The attack was accompanied at times by momentary and very slight confusion, but I never observed that it was followed by any symptom whatever. I could sometimes determine an attack by pressing on the globe of the eye, and I discovered accidentally that I could bring back the sight very soon, by bending the head between my legs, so that the capillaries of the face got flushed."

(b). Bilateral amblyopia or amaurosis may occasionally follow certain epileptic attacks, and the loss or impairment of vision in these cases may last for hours, days, or more rarely even for several weeks. Again, in the allied condition 'migraine,' it often happens that the attacks are immediately preceded by a peculiar disturbance of vision, long known, but to which Ross has lately given the appropriate name of 'scintillating scotoma.'

He gives the following condensed description of this condition, and of his own experience of it:—"This form of disordered vision appears at the onset to consist of a scotoma, generally affecting both eyes, though not to the same extent. The loss of sight is limited at first to a small portion of the visual field, which may be centric or eccentric; but it soon spreads and as a rule one lateral half of the field of vision is affected. At other times the affection of vision is central, and images of surrounding objects to which the axes of the eyes are not directed may be visible as usual. The blindness is accompanied by spectral appearances, which as a rule become gradually developed as the black spot extends. In their simplest form they consist of a luminous border surrounding more or less completely the blind area, and widening as the latter expands (Lieving). This luminous circle or arc is subject to a rapid oscillatory movement, which has been variously described by different observers. In the more pronounced form of the affection the luminous border assumes a zigzag outline which has been compared to the angles of a fortification. It is also fringed by gorgeous colours, which are in continual trembling movement, or appear to 'corruscate' or to emit a 'shower of sparks.' The phenomenon generally lasts from a quarter to half an hour, and then gradually passes off. The few times that I have experienced this phenomenon I could only compare the luminous arc to a horseshoe rainbow with its convexity directed upwards, and without either zigzag outline or oscillatory movement. I could always see objects clearly in the lower half of the field of vision, and the partial blindness passed off in less than five minutes, without being followed by any other symptom."

Reflex vaso-motor spasms affecting either some of the optic centres or the optic tracts or nerves, may be the cause of amblyopia or amaurosis in certain cases, especially when some source of irritation exists in the course of one or other of the branches of the trigeminus, either from carious teeth, tumours or periosteal thickenings in the foramina or canals through which the branches pass, or even occasionally from exposure to cold. It is said, moreover, that amblyopia or amaurosis of a similar type may be produced by irritants in the stomach or intestines, or by some abnormal conditions of the uterus or other abdominal organ, though it is altogether more doubtful how far such conditions may be capable of producing these results. The amblyopia or amaurosis in all this class of cases is apt speedily to disappear on the removal or decided mitigation of the original source of irritation.

Certain toxic agents consumed or taken for some time in small yet excessive quantities, are apt, in some obscure way, to induce amblyopia or amaurosis. Among the principal of such agents are lead, alcohol, and tobacco.

Again amblyopia or amaurosis may supervene, sometimes rather suddenly, during convalescence from many acute diseases, the mode of production of which is also extremely obscure. Such conditions are most apt to occur after typhus fever, scarlet fever, measles, and other such affections.

Lastly we come to a heterogeneous group of conditions, characterized mostly by exhaustion or debility, in which temporary amblyopia or amaurosis is also apt to manifest itself. This class is composed of cases in which profuse hæmorrhages from the stomach, the womb or any other part, have been of frequent occurrence; or it may include cases of venereal excesses, or cases in which some profuse discharges have been suddenly stopped. We may have to do here with effects produced, either by poverty of blood and anæmia, or, perhaps, speaking more specifically, by reason of some vaso-motor spasms or irritability of the vessels supplying some optic centres or tracts, which such a general health-state is prone to favour.

(2).—We must now turn to cases in which Amblyopia and Amaurosis are associated with decidedly abnormal conditions of the optic discs.

These are the instances in which there is the association of **Optic Neuritis** or **Optic Atrophy**, the conditions of origin and stages of which we are about to consider in the following sections. Such changes are the consequences, so far as the optic discs are concerned, of actual disease or of abnormal conditions of various kinds operating in or upon the optic nerves.

We have, therefore, now to consider the amblyopia or amaurosis which may be associated with organic disease of the optic nerves, just as in the preceding section we considered the similar defects of vision, that may be met with in the absence of any such disease.

LESIONS OF THE OPTIC NERVE.

In order to obtain an accurate knowledge of the existence and nature of organic disease of the optic nerve, during life, we have to study any changes from its normal condition that may be seen at its entrance into the eyeball, by means of the ophthalmoscope. A correct interpretation of the results of such examinations will be facilitated, and the subject of optic neuritis and optic atrophy made plainer, if we, in the first place, briefly recall to the student's mind certain anatomical details concerning the envelopes of the optic nerve, and the mode in which its fibres spread out so as to form the retina.

The Envelopes of the Optic Nerve are composed first of an external or dural sheath, which is a thick fibrous envelope continuous with the dura

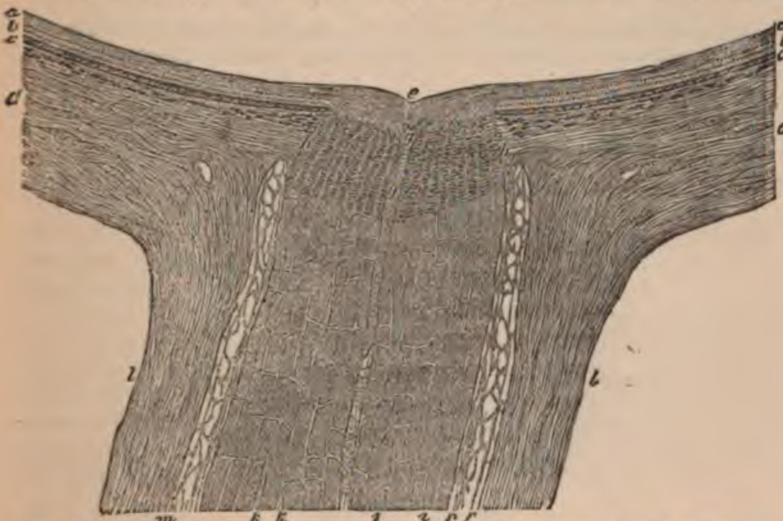

FIG. 90. HORIZONTAL SECTION THROUGH THE OPTIC NERVE AT ITS POINT OF INSERTION INTO THE GLOBE, SHOWING ITS PASSAGE THROUGH THE MEMBRANES OF THE EYE [after Landois].

a, Internal; *b*, external layers of the Retina. *c*, Choroid. *d*, Sclerotic. *e*, Physiological cup. *f*, Central artery of the retina; *g*, point of its bifurcation. *h*, Lamina cribrosa. *l*, Dural sheath. *m*, Sub-dural space. *n*, Sub-arachnoidal space. *r*, Arachnoidal sheath. *p*, Pial sheath. *i*, *i*, Bundles of nerve fibres. *k*, *k*, Connective tissue trabeculæ.

mater ; secondly, of a middle sheath continuous with and resembling the arachnoid—the so-called arachnoidal sheath; and thirdly of a continuation of the pia mater—the pial sheath.

Between the dural and arachnoidal sheaths is a lymph space, freely communicating with the sub-dural space of the brain; while between the arachnoidal and pial sheaths there is another but quite separate lymph space, communicating freely with the subarachnoid space of the brain—and known as the subarachnoid space of the optic nerve.

The substance of the **Optic Nerve,** as shown in the figure, is made up of multitudes of bundles of nerve fibres, isolated from one another by a connective tissue framework, in which are contained the vessels that supply the nerve. This connective tissue framework also contains minute lymph spaces continuous with the large lymph space beneath its pial envelope.

In the axis of the nerve is lodged the arteria centralis retinæ.

The fibres of the optic nerve at the back of the eyeball pass through the sclerotic and choroid membranes, though not in a similar manner. The bundles of the optic nerve pass through a number of sieve-like apertures in the sclerotic coat, and this perforated portion of the coat (Fig. 90, *h*) is known as the lamina cribrosa. In the case of the choroid coat, however, there is an actual round or oval opening, named the choroidal ring through which the optic nerve passes.

Ophthalmoscopic Appearances of the Healthy Fundus.—It will be useful to call attention to a few of the characters presented by the fundus of

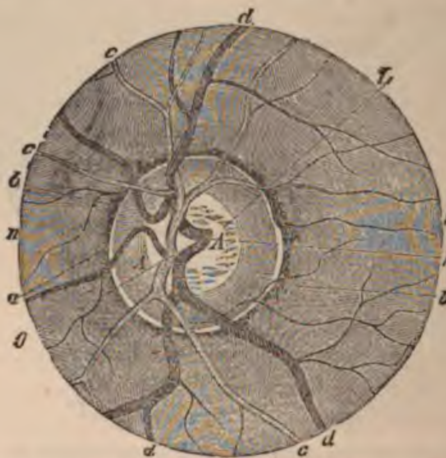

FIG. 91. REPRESENTATION OF THE FUNDUS OF THE HUMAN EYE [Landois after Jæger].

A, A, Optic papilla. *a*, Sclerotic ring. *b*, Choroidal ring. *c*, Arteries. *d*, Veins. *g*, Point of bifurcation of the central artery of the retina. *h*, Point of bifurcation of the central vein. L, Lamina cribrosa. *t*, Temporal, and *n*, nasal side of the fundus.

the healthy eye though the student is referred to the excellent 'Manual and Atlas of Medical Ophthalmoscopy' of Dr. Gowers, as the most accessible work in which he may find a large number of illustrations of the various stages of optic neuritis and optic atrophy. The descriptions given by Gowers of these conditions are here closely followed.

As the fibres of the optic nerve curve away from the lamina cribrosa and over the edge of the choroid they rise slightly above the surrounding level of the retina; and this slight prominence of the termination of the optic nerve has caused it to be commonly spoken of as the optic papilla.

The optic papilla is, however, marked by a central depression, known as the physiological cup (Fig. 90, *e*), produced by the fibres radiating away from the centre over the edge of the choroid.

The choroidal aperture is generally slightly larger than the termination of the nerve, consequently a narrow ring of the sclerotic coat is commonly to be seen immediately around the optic disc, which is known as the **sclerotic ring** (Fig. 91, *a*).

In regard to the **optic disc** Gowers says:—"In the optic disc we have presented to view the termination of a nerve—a structure consisting of nerve fibres, a little supporting connective tissue (especially abundant around the central vessels), and a number of blood vessels, for the most part capillaries, which confer on the disc its tint. The nerve fibres radiate and spread out in the retina, but not equally on all sides, being few on the temporal side, towards the macula lutea, and numerous on the nasal side and especially above and below. The minute vessels of the disc are derived partly from the posterior ciliary (choroidal) arteries, and partly from the central retinal artery. . . . The tint of the circumferential portion of the disc is, as already explained, deepest where the nerve fibres are most numerous, and hence the nasal half of the disc is naturally much redder than the temporal half. The arrangement of the nerve fibres also causes the side of the central cup to be steep on the nasal and shallow on the temporal side, the difference being proportioned to the inequality with which the nerve fibres are distributed. When the fibres are almost all packed on the nasal side, the cup may be very large, and extend on the temporal side to the margin of the disc. Often, however, there is no paler central cup."

The central artery of the retina is a branch of the ophthalmic artery, and the retinal veins empty themselves almost completely into the cavernous sinus. The exact branching and distribution of these vessels is subject to considerable variation in different individuals, but about the most common arrangement is that represented in Fig. 91. The disc itself is supplied with blood partly by twigs coming off from the central artery of the retina, but partly also by others derived from the posterior ciliary arteries.

The effects produced by disease in the various optic centres and in the optic tracts as far as the commissure, have already been considered (pp. 134-150). We have now briefly to consider the conditions which give rise to disease of the optic nerve itself in any part on the distal side of the commissure, and more especially the effects of such disease upon the papilla as revealed by ophthalmoscopic examination.

The changes in the optic discs belong to two principal categories. In the one we have to do with conditions of congestion or inflammation, associated with more or less swelling of the substance of the discs. These states present many variations in degree, but generically are known as 'optic neuritis.' In the other, we have states of diminished vascularity, together with wasting of the substance of the disc: conditions, again, subject to much individual variation, but generically included under the term 'optic nerve atrophy.'

Gowers summarises the principal forms of these conditions as follows:—

A. *Morbid states of the optic nerve, characterized by increased vascularity or signs of inflammation.*

1. Simple congestion of the disc; undue vascularity, redness, the edge softened, but not obscured, and no swelling.
2. Congestion with œdema of the disc (slight neuritis or papillitis); increased redness with slight swelling; obscuration of the edge of the disc, complete to the direct examination, incomplete to indirect examination.
3. Neuritis, or papillitis; increased redness and swelling, with obscuration of the edge of the disc, complete in degree, partial or total in extent.

B. *Diminished vascularity and signs of wasting.*

1. Simple atrophy; increased pallor from the first; 'primary atrophy.'
2. Congestive atrophy; secondary to congestion; pallor slowly succeeding simple congestion.
3. Neuritic atrophy, succeeding pronounced neuritis; 'consecutive atrophy,' or 'papillitic atrophy.'

Only a few words can be said here concerning the varied characteristics presented by these different states in different individuals, in accordance with the fluctuating conditions of their origin, and the various stages of their development or retrogression.

A.—OPTIC NEURITIS.

The colour of the optic disc is subject to considerable variation in different individuals, within the limits of health. Distinct difference of tint between the two discs of a person under examination is always a fact of some significance; but where this does not exist, we should, as Gowers points out, look especially for two characters, the existence of which may enable us to affirm the presence of a pathological increased vascularity. These are, first, the extension of the redness so as to invade the physiological cup, and perhaps to obscure it altogether; and secondly, the fact that pathological redness has a tendency to render the sclerotic ring or the edge of the choroid indistinct—with the result of blurring the sharpness of outline of the disc.

Thus uniformity of colour over the disc, together with an indistinct or softened edge are the most reliable characteristics of slight pathological hyperæmia.

Optic neuritis itself is divisible into two stages, or two degrees of intensity. Slight neuritis or **congestion with œdema** is characterized by increased redness, swelling, and cloudiness—masking the edge of the disc to 'direct' but leaving it perceptible to 'indirect examination.' The increased vascularity of the disc may present a striated appearance at the periphery; the disc is, however, commonly redder in the centre, owing to the smaller amount of central swelling causing less obscuration of the vessels in this situation.

The subsequent stage, or condition of greater intensity, is known as **neuritis** or **papillitis.** This is to be met with in various developmental stages, but in all the swelling and opacity are sufficient in amount to conceal the edge of the disc both to direct and to indirect examination—so that the transition from the disc to the retina is gradual—the edge being 'blurred.' The disc becomes redder (similar in colour to that of the adjacent choroid) and more swollen, whilst the striation of the periphery also becomes more distinct. At times the disc, especially towards the centre, is of a greyish red colour, and all trace of the physiological cup has disappeared. The swelling and opacity may be uniformly distributed, or it may be much more distinct on the nasal than on the temporal side of the disc. Minute hæmorrhages may also occur at this period either on some part of the disc or at its confines. The arteries may now be a little more difficult to recognize, owing to their similarity in colour to that of the disc, and may be somewhat concealed at their point of emergence, but commonly present no distinct alteration at this stage. The veins may or may not be dilated, and they appear dark as they pass down the sides of the swelling.

In later stages the swelling increases, the arteries may become narrowed and more obscured and the veins still more dilated. The swelling increases laterally as well as in depth, so that the swollen and vascular area may after a time be even two or three times the diameter of the optic disc itself. A large number of cases proceed no further than this, and, as Gowers points out, even a neuritis which has attained such a stage may clear completely, the inflammatory products being for the most part removed, and those which remain merely causing a little increase of tissue in the middle of the disc. White lines along the vessels may also persist as remnants of the previous inflammation.

More extreme degrees of change are, however, often met with, in which the swelling increases both in depth and laterally and where signs of strangulation of the vessels occur—causing the arteries to be distinctly diminished in size, and the veins to become more dilated and tortuous. This extreme form is often spoken of under the name of 'choked disc' (Allbut)—a substitute in this country for the term 'stauungs-papilla' (Von Graefe).

It is contended that even such an extreme form of so-called optic neuritis may be produced merely as a result of mechanical congestion; and if that be true, of course the minor manifestations of optic neuritis might be similarly induced. Others hold that a similarly extreme form of the disease may be induced by a mere extension in degree of the inflammatory process, which they believe to be the efficient cause of optic neuritis in its less developed stages. It seems, moreover, to be commonly admitted now, by many excellent observers, that no really distinct and trustworthy criteria exist by which the 'choked disc' may be safely distinguished from the so-called 'descending neuritis' in its later stages.

In the 'choked disc' stage the papilla becomes much more prominent and broader. The form of the swelling varies. It may remain conical, though usually the sides become steeper and the top more or less flattened. The sides may even overhang so that the tumour has a fungiform shape, and the vessels as they pass over the side may be concealed by the edge of the swelling and reappear in the fundus in a different position.

The arteries are much narrowed, but often more or less hidden in the substance of the swelling, so that they may appear first in the retina near its edge. The veins may be also greatly hidden in the substance of the swelling, though some of them are usually visible towards its edge. The veins are greatly distended and hæmorrhages are common, not unfrequently occupying the overhanging edge of the swelling. Beyond the edge of the disc the veins usually present many curves and twists. "The tint of the strangled swelling is usually a full red, mottled and streaked from enlarged vessels and small extravasation, the striation due to the nerve fibres is commonly lost. The retina adjacent is often the seat of hæmorrhages which may extend along the vessels from the disc. . . . Sight, if not previously lost, fails entirely, and often very rapidly during the stage of strangulation. The time taken for the development of these changes varies within wide limits. A neuritis may remain for months and even years in the slighter stage, or most intense strangulation may be developed in a few weeks" (Gowers, *loc. cit.*, p. 46).

In different cases of optic neuritis, the pathological conditions may be arrested at any stage of their evolution; may remain stationary at such stage for variable periods; and then, spontaneously or under the influence of remedies, may gradually subside so as more or less completely to disappear. For stages anterior to those in which strangulation occurs, this disappearance at times of morbid changes may, as above stated, be almost complete. But where optic neuritis has been developed to such an extent as to have attained the 'choked disc' stage, the process of subsidence is slower, always less perfect, and is much more prone to lapse into a condition of optic atrophy. When subsidence begins to occur, the distension of the veins after a time grows less, the redness diminishes, hæmorrhages cease, previous extravasations gradually disappear, the swelling lessens in all directions, and if it has been fungiform it again becomes conical. The commencing pallor shows itself first in the highest part of the swelling and slowly spreads downwards and outwards towards its margin. The centre of the swelling soon presents a distinct depression, from which the arteries emerge often bridged over by cicatricial tissue. The veins are more distinct and usually less concealed, except just beyond the edge of the disc so long as swelling still exists. As the white area narrows to near the limits of the disc, the edge of the choroid and sclerotic appear more and more distinctly. The disc now has a white appearance, and for a long time presents no central depression. At a later stage still, when all swelling has completely disappeared, the disc generally assumes a slightly greyish tint, especially to direct examination. There may be a zone of slight choroidal atrophy, giving the disc an apparently irregular outline. At times the process of subsidence occurs in an irregular manner, clearing away from some parts of the disc much more rapidly than it does from others. Hæmorrhages that may have occurred into the retina near the margin of the swollen disc, during the period of its subsidence become absorbed; or they may leave as remainders spots of pigment, or white spots of degenerative origin.

In regard to the *symptoms* of optic neuritis, it is a fact of great importance long insisted upon by Hughlings Jackson that in many cases these may be entirely absent. Gowers also says :—"The degree of neuritis which may exist with no impairment of acuity of vision, is remarkable." He adds, "In more intense cases however sight is impaired or lost, and this constitutes the chief symptom of optic neuritis. Photophobia may occur in simple congestion, but is practically unknown in neuritis. Pain in the eye is very rare. Pain in the head may occur in cases of apparently idiopathic papillitis. It is of course a very common accompaniment of symptomatic inflammation, but is then generally to be accounted for by the intra-cranial disease."

In the cases where vision is affected it often fails in one eye before the other, and may make rapid progress, so that sight may be completely lost in the course of a few days. At other times the impairment of vision is much slighter and advances much more slowly. Restriction in the visual field generally coexists with diminution in the acuity of vision. There may be peripheral contraction of the visual field to a very variable extent, either in a regular or very irregular manner (Fig. 92). At times this kind of change may coexist with hemiopia, as another consequence of the intra-cranial disease. A central scotoma is rare from this cause and is said to occur only where a lesion exists at the yellow spot.

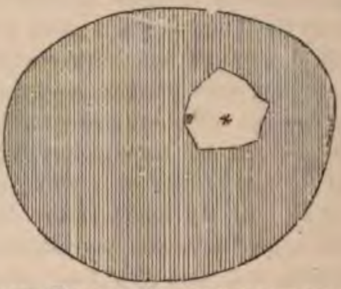

Fig. 92. Diagram of the Field of Vision in a case of subsiding optic neuritis in the left eye. Case of Cerebellar Tumour [after Gowers].

The outer boundary of the figure is the limit of the average normal field. Vision was lost in the shaded area, preserved only within the inner line around the fixing point, the position of which is indicated by the asterisk.

Colour vision is not unfrequently impaired in optic neuritis, and that too, at times, as Galezowski pointed out, even where there is no diminution in the acuity of vision. The order in which the loss

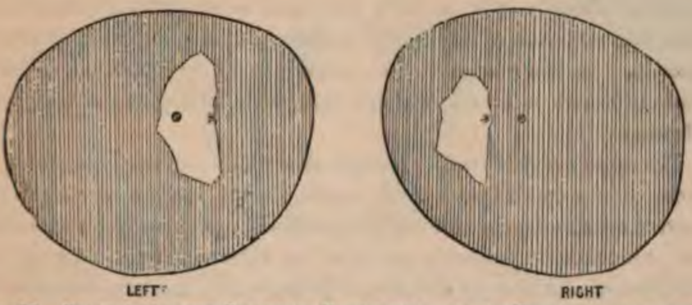

FIG. 93. DIAGRAM OF THE FIELDS OF VISION IN A CASE OF HEMIOPIA AND DOUBLE OPTIC NEURITIS [after Gowers].

The star represents the fixing point, the dot the position of the blind spot. The outer boundary of the shading is the normal limit of the field, the shading the area in which sight was lost. There is seen to be loss of the whole right half of each field with concentric limitation of the right halves.

occurs is often irregular, though at other times it follows the rule already indicated (p. 388).

The amblyopia occurring during the height of the inflammation may lessen as the inflammation subsides, and be again increased later on by the contraction of the new connective tissue compressing the nerve fibres. In other cases, where there has been no affection of sight during the inflammatory stage, it may become seriously impaired in the manner above indicated during the late stages of subsidence. In the extreme cases optic atrophy with amaurosis occurs.

By far the most common *cause* of optic neuritis is intra-cranial disease of some kind, and of these tumours take altogether the first rank in point of frequency, and that to a considerable extent irrespective of their size or seat. After these come basal meningitis, and abscess. It is very rarely associated with cerebral hæmorrhage, though it occurs rather more frequently with softening of the brain due to thrombosis or embolism of a large vessel. In almost all such cases the optic neuritis when it occurs is double, though it may be unequal in its development on the two sides.

It is most important, however, to bear in mind that double optic neuritis may be met with in chronic Bright's disease, and also in chronic poisoning from lead, because in these conditions, and especially in the former, it may coexist with headache, sickness and other symptoms, which might, but for the recognition of the Bright's disease, and in the absence of other ophthalmoscopic signs of this condition, raise a strong suspicion of the existence of an intra-cranial growth. Double optic neuritis may also be met with in association with one or other of the acute specific diseases, or with profound anæmia. In some cases it has seemed to follow exposure to cold; while in others it occurs without assignable cause, as an idiopathic affection.

The optic neuritis itself presents essentially similar characters, in these various conditions. And, again, though various views are put forward concerning the actual pathogenesis of optic neuritis, and though the condition is probably produced at different times, now by one mechanism and now by another, it seems to be generally conceded by the best observers, that such differences in mode of pathogenesis cannot be discriminated with any certainty by ophthalmoscopic examination.

Unilateral optic neuritis is most apt to be caused by tumours or inflammations involving the posterior part of the orbit. It is an extremely rare event for one eye only to be affected under the influence of one of the general conditions previously referred to.

Nothing more than a mere mention will be made here of the different views that have been advanced in order to account for the pathogenesis, or actual production of optic neuritis, as this is a subject concerning which much uncertainty still exists.

(1).—A 'descending neuritis' (Von Graefe)—that is, an inflammation communicated to the optic nerve from an inflamed meninges—is commonly regarded as one of the modes in which optic neuritis becomes established.

(2).—An increase of intra-cranial pressure is supposed in other cases, in the absence of any inflammation of the trunk of the optic nerve itself, to lead to *mechanical congestion* of the optic disc, and thus to the production of all the signs met with in optic neuritis. Two versions of this view have been advanced.

> *a.*—The increase of intra-cranial pressure causes pressure upon the cavernous sinus and thus impedes the return of blood from the optic disc, producing a mechanical congestion thereof (Von Graefe).
>
> *b.*—The increase of intra-cranial pressure (owing to the fact that the subvaginal space round the optic nerve is continuous with the subdural space around the brain) tends to distend the sheath of the optic nerve with fluid (Fig. 94), and thus gives rise to an undue amount of pressure upon the optic nerve fibres and upon its vessels—the fluid penetrating even into the lymph spaces of the nerve and thus more effectually exerting pressure upon the several fibres of the nerve and upon its vessels, thereby again entailing mechanical congestion (Schmidt).

(3.)—In cases of cerebral tumour, these are supposed to act like foreign bodies in producing 'irritation,' which affects certain tracts of grey matter, and these again are supposed to send reflected incitations along the vasomotor nerves on the optic vessels, leading to frequent contractions followed

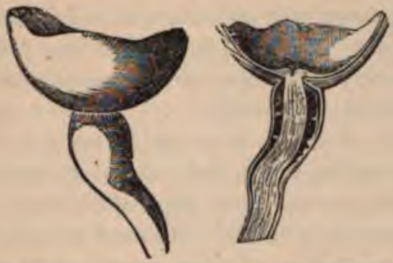

FIG. 94. POSTERIOR SEGMENT OF EYEBALL AND OPTIC NERVE, SHOWING DISTENSION OF THE SHEATH OF THE NERVE AND SWELLING OF THE PAPILLA. FROM A CASE OF CHRONIC TRAUMATIC MENINGITIS. NATURAL SIZE [after Pagenstecher and Genth].

by paralyses, and, as consequences, the trophic changes which constitute the condition known as 'optic neuritis.' This is commonly described as the 'vaso-motor' theory (Schneller, Hughlings Jackson, and Benedikt).

Neither of these agencies can be supposed to be effective in all cases; sometimes we may have to do with one of them and sometimes with another; at other times two or even three of such agencies may coexist and act in combination. An immense amount of difference of opinion still exists on the subject of the pathogenesis of optic neuritis, into which, however, it would be unsuitable to enter further in this work.

In the *diagnosis* of optic neuritis itself three characters have to be especially attended to, viz., (*a*) the amount of *redness* of the optic disc, (*b*) the degree of *obscuration of its edge*, and lastly (*c*) the amount of *swelling* of the disc.

(*a*). The normal variations in the amount of redness, and the characters that should be regarded as unnatural, have already been referred to (p. 396).

(*b*). Obscuration of the edge of the disc when present is a character of great significance. It is due to opacity of the layer of nerve fibres lying in front of the edge of the disc, when they have become altered and rendered more opaque by disease. It should be borne in mind, however, that a slight amount of obscuration of the edge may be present normally in those situations in which the layer of healthy nerve fibres is naturally thickest, that is on the nasal side (and especially when the central cup is very large), as well as above and below along the course of the great vessels. In these situations, also, where the nerve fibres are thickest, there may, even in conditions of health, be some appearance of a radiate striation; where optic neuritis exists, however, such striation is more frequently met with all round the disc, and is caused in part by pale lines from altered nerve fibres and in part by red lines due to an increase in fine vessels lying between the nerve fibres (Gowers).

(*c*). The last characteristic to be sought for is the existence of an undue amount of swelling of the optic papilla. Normally the surface of the papilla is a little anterior to the plane of the retina, and only slight variations are met with in health. But such elevation is rarely sufficient to be appreciated even by the most careful ophthalmoscopic examination. As a rule, a prominence which is readily recognizable is pathological. In optic neuritis numerous degrees of elevation may be recognized by resorting to the following well-known expedients. When the 'direct method' of examination is being employed the elevation of one object in the fundus above the level of an adjacent object (as of a vessel on the edge of the physiological cup above a vessel at its bottom) is to be appreciated by the observer moving his head from one side to the other, as far as possible without losing sight of the objects. The alteration of the relative position of the objects during this manœuvre will be proportioned to the difference in level —and thus, to the amount of swelling existing. When the 'indirect method' is being employed the same kind of estimation may be made by means of a lateral or vertical movement of the lens, which produces the same effect as a corresponding movement of the observer's head.

B.—ATROPHY OF THE OPTIC NERVE.

In this condition we have to do with diminished vascularity and more or less wasting of the optic papilla—conditions just the reverse of those met with in optic neuritis, of which optic atrophy is often a sequence.

Regarded from the mere point of view of ophthalmoscopic appearances, optic atrophy may, as we have seen, be divided into three categories, under which the great majority of cases may be included, viz. (a) simple atrophy, (b) atrophy following congestion, and (d) atrophy consecutive to neuritis. The distinction between the first and second categories is, however, probably of little value, since where atrophy originates under apparently the same conditions it may sometimes manifest itself without, and sometimes with, a preliminary period of more or less marked congestion. The atrophy in these cases is, moreover, not supposed to be a consequence of the initial congestion;

while in the cases included under the third category, the atrophy is regarded as a direct consequence of the pre-existing neuritis.

For our present purpose it will be better to divide cases of optic atrophy into two categories (1) primary atrophy of the optic nerve, and (2) secondary atrophy of the optic nerve.

(1). **Primary atrophy of the optic nerve** occurs in its most typical form as a forerunner of locomotor ataxy or, in a certain number of cases, concurrently with the development of the disease. It may occur also, but much more rarely, in association with general paralysis of the insane, or disseminated sclerosis. Probably the latter cases ought to be included under the head of secondary atrophies, since, in the rare cases of this disease in which optic atrophy occurs, the atrophy of the disc would most likely be produced by an extreme overgrowth of connective tissue (patch of sclerosis) in some part of the optic nerve behind the eyeball. In the cases of locomotor ataxy and of general paralysis of the insane, however, in which optic atrophy occurs it is much more likely to be due to a primary and independent change occurring in the optic nerve elements themselves, leading to a secondary overgrowth of the connective tissues of the disc.

The remainder of the cases of primary atrophy of the optic nerve constitute, from an etiological point of view, a very heterogeneous assemblage of conditions under which, and for the most part very rarely, optic atrophy at times presents itself. In almost all the cases, nothing is known as to the actual pathogenesis of the atrophy in these different conditions, and, looking to the rarity of the associations, it may be even difficult to say whether we have to do with a mere coincidence or with a real causal nexus. Among these alleged causes, we find menstrual disturbances, sexual excesses, migraine, certain fevers (intermittent, typhoid, etc.), gastro-intestinal affections, syphilis, diabetes, exposure to cold, various toxic agents (such as alcohol, tobacco, and lead), as well as blows in the neighbourhood of the orbit or on the eyeball. Lastly, it is worthy of note that this primary atrophy seems to be sometimes hereditary: it has been known to affect all the males of a family soon after puberty.

The appearances that present themselves in the optic discs seem to be essentially similar in all these cases so that we may now refer (1) to the ophthalmoscopic characters of simple or primary atrophy of the optic disc.

Atrophy of the nerve fibres of the optic disc is accompanied by a diminution in size or disappearance of many of its small vessels and capillaries—the result being that the disc becomes more pallid than natural, and that it shrinks or becomes more or less 'excavated,' although its actual area remains the same.

Where the condition is pronounced it is one that is readily recognized; but in regard to its earlier stages mistakes are easily made, and doubts are not unfrequently felt as to the existence or not of optic atrophy, especially where the observer's experience is small. In such cases, proper precautions should be taken in regard to the mode of examination, and certain qualifying conditions, to which reference will now be made, should also be borne in mind. We shall here, again, closely follow the account given by Gowers, referring successively to the characteristics to which particular attention must be paid, in coming to a conclusion whether optic atrophy exists or not in any given case that may be under examination.

Pallor.—In order to form a correct estimate as to the colour of the disc it is important to examine it with a weak illumination and by the 'direct method,' so as to allow of the reflection of as little light as possible. In a strong light a faintly tinted object will appear white; hence the importance of employing a weak illumination in order to recognize a slight colouration.

The normal variations in the colour of the optic disc have already been referred to (p. 395). It is, further, of importance, in estimating the slighter degrees of atrophy, to bear in mind that, as a rule, the disc becomes paler as life advances, and a slight grey tint is mingled with the red, though the latter is still perceptible. The physiological cup, if slight, is often indistinct late in life. Thus a tint which is normal in the old would be suggestive of atrophy in the young. Although in anæmia the disc becomes paler, yet the change of tint from this cause is generally insignificant in comparison with its normal variations in colour.

When a pathological pallor of the disc is pronounced it extends over its whole area; but a commencing pallor is most apt to show itself in that part of the disc which is normally palest, that is, on the temporal side where the nerve fibres are least numerous. The change in this part is, however, of most significance in those cases where, owing to the small size of the physiological cup, the temporal half of the disc normally possesses a distinctly vascular tint. It has little or no significance in that large number of cases in which the physiological cup is large and slopes gradually to the temporal margin of the disc, because in such cases this portion may be naturally as pale as in advanced atrophy.

For the above-mentioned reasons, the part on which attention should be principally fixed is that which normally presents the most vascularity, viz., the nasal portion of the disc. Here, when patients are examined from time to time during the development of optic atrophy, the tint may be seen to become gradually paler, the red sometimes simply fading and leaving a white colour in its place; in other cases a grey becomes mingled with the red, and gradually preponderates as the red tint fades, till ultimately a pure bluish or greenish-grey colour is left. These two varieties constitute, in their extreme types, the white and grey forms of atrophy respectively. Intermediate conditions, of course, also exist, and to the 'direct method' of examination some grey tint may always be distinguished, even in discs which appear of tendinous and even chalky whiteness when examined by the indirect method. This grey mottling tends to increase as time goes on. The atrophy leaves the edge of the disc very distinct and sharp. The sclerotic ring is clearer than it is naturally, but may not at first be recognized by the 'indirect method,' because it is not easily differentiated from the white surface of the altered disc. This sharpness of the edge is due, in part, to the fact that the choroid presents its normal characters up to the margin, and thus gives to the clear outline a sharp-cut aspect, which is the peculiar characteristic of 'simple atrophy.'

Excavation.—In some cases of simple atrophy the depression of the disc is considerable, and in others it is slight or absent. The amount of the depression is always proportionate to the wasting of the nerve trunk; and the degree of wasting in the trunk is variable, owing to the fact that in this as in all cases of nerve atrophy we have to do with two processes—first, a wasting in the nerve fibres, and secondly, an overgrowth of the connective tissue between these nerve fibres. Where the wasting is great and the overgrowth small we should expect distinct excavation; on the other hand, where the overgrowth of connective tissue is excessive, it may fully occupy the space yielded by the wasted nerve fibres, and then there would be no unnatural excavation of the disc. In grey atrophy, the shrinking of the nerve and excavation of the disc is slight, because this is the form in which most connective tissue is developed.

When pathological excavation exists with atrophy, its special character is that it affects the whole disc, and commences at the sclerotic ring. It may often be recognized by the change of level of the retinal vessels at this region, a change which becomes most distinct on lateral movement of the observer's head. This is an important diagnostic sign, because normally the depression of the centre of the disc never begins at the sclerotic ring, except in some of the cases in which a very large physiological cup exists, and even then the depression begins at the edge only on the temporal side of the disc.

The mottling of the lamina cribrosa may become very distinct at the bottom of the excavation. Where the normal cup was large, the excavation may reveal the lamina cribrosa in almost the whole extent of the disc, the grey mottling corresponding to the bundles of degenerated nerve fibres, the white intervals to the meshes of the lamina.

The Retinal Vessels.—In some cases of simple atrophy the retinal vessels become reduced in size, in others any such reduction is scarcely appreciable. When present, the diminution in size is commonly to be observed first in the arteries and subsequently in the veins. Why they should shrink in some cases and not in others cannot be said to be definitely known. In cases in which there is a retro-ocular neuritic process, it might be expected that shrinking would occur.

In the cases in which **atrophy is preceded by a stage of congestion,** the disc has, at first, a dull red tint, with a soft-looking surface, the redness being uniformly distributed over the disc. The edges of the disc are less sharply defined than in health; they are visible, but are softened. The congestion may persist for a long time; but after a variable period the disc slowly becomes paler, and ultimately a condition of greyish white atrophy is reached. Occasionally the stage of congestion may be associated with slight œdema, leading to some amount of swelling of the disc for a time.

(2). **Secondary Atrophy of the Optic Nerve** may be caused by morbid processes in very different sites, and to some of these our reference may be very brief; partly because the physiological relations upon which they depend have been already referred to (p. 137), and partly because much uncertainty still exists in regard to the amount of atrophy that follows lesions in some of the sites now to be referred to.

(a).—Lesions in the region of the angular or supra-marginal gyrus, or of the external geniculate body, are supposed by some to be capable, after a rather long interval, of causing atrophy of the opposite optic disc, and, to a less extent, of the outer half of the disc on the side of lesion. Both Gowers and Bernhardt think they have seen cases belonging to such a category.

(b).—A destructive lesion involving one optic tract gives rise to bilateral symmetrical hemiopia, but this visual defect, even after some years, is associated with no appreciable atrophy of the affected halves of the disc. At most there may be some amount of pallor. On this subject, Gowers says:—"Some observers have described an ultimate slight pallor of the corresponding halves of the discs, but I have never been able to trace this in any of the cases of hemiopia (about thirty) in which I have carefully looked for it." These positive statements concerning the effects of lesions in the optic tracts seem to me to inspire considerable doubt in regard to the existence of the atrophy supposed to be caused by lesions of the optic centres referred to in the previous paragraph.

(c).—Pressure on the optic chiasma, is said to be a common cause of optic atrophy without the intervention of neuritis. The pressure may be brought about by tumours springing from any of the adjacent parts of the brain or cranium, or by aneurysms of adjacent arteries. Hydrocephalus is another cause; the distended third ventricle pressing directly upon the chiasma. In some cases of meningitis, again, although inflammation may extend to the nerve and cause some amount of neuritis, it may also at times bring about such an amount of pressure upon the chiasma as to cause atrophy of the optic nerves and consequently of the discs. In some of these cases the atrophy of the disc is apt to be partial, or more marked in one than in the other eye.

(d).—The optic nerves themselves (one or both) may be damaged in any part of their course from the chiasma to the eyeball, to such an extent as to lead to atrophy of the optic disc. Damage to the optic nerves in front of the chiasma may be caused by tumours, exostoses, or meningitis. This may be affected within the optic foramen by thickening of the dura mater in that region, or by rheumatic or syphilitic thickening of the bone itself. Various morbid processes, again, may involve the optic nerve at the back of the orbit, in such a way as to lead to atrophy of one optic disc—for in such cases the atrophy is often single. Patches of sclerosis, occurring in association with disseminated sclerosis, may, when well developed, after a time entail atrophy of one or both optic discs.

It is important to bear in mind that in all the cases of secondary atrophy due to disease in the chiasma or in the optic nerves to which we have just been referring, the appearances in the discs themselves may not, and often do not, differ in any appreciable way from those that are passed through during the development of simple or primary atrophy—stages which have been fully described. It has been already stated, also, that 'retro-ocular neuritis' may, in some cases, lead to an atrophy which is preceded by a stage of congestion with or without slight œdema, and that in these cases the narrowing of the vessels is apt to be most marked, and ultimately may approach in degree that met with in the atrophy about to be considered, viz., that which is consecutive to intra-ocular neuritis. In other cases of so-called primary atrophy, however, an initial process of congestion may be met with; while, on the other hand, in some cases of 'retro-ocular neuritis' no initial stage of congestion precedes the development of the atrophy.

(e).—Lastly we may have optic atrophy secondary to certain **morbid processes occurring within the eyeball,** such as (α) intra-ocular neuritis, (β) embolism or thrombosis of the central artery of the retina or of one of its branches, (γ) choroido-retinitis, or (δ) as a sequence of blows upon the eyeball.

Of these, the most frequent and by far the most important form of atrophy is that which is secondary to optic neuritis, commonly known as (α) *papillitic or consecutive atrophy,* and to this form we shall first direct the student's attention.

As already indicated, optic neuritis may clear away so completely as to leave no trace behind it; at other times it may leave certain slight pathological traces of its previous existence, though not of a kind to interfere with vision; in a third set of cases (those to which we are now about to refer) it leaves behind it very marked pathological changes together with loss of vision or amaurosis.

In the cases where atrophy follows optic neuritis, many of the inflammatory products, instead of being absorbed, become converted into connective tissue, and at the same time the nerve fibres become more and more destroyed. During the process of subsidence, the pale swelling left by the inflammation gradually diminishes both in depth and in area. After a time it becomes lowered to the level of the surrounding retina, and confined within the limits of the disc itself, where its margin gradually becomes more and more sharply defined. But as the swelling recedes from the edge of the choroid, it is often seen that the edge of the latter has been damaged irregularly by an atrophic process, with the result of apparently giving the disc a very irregular outline. The presence of the remaining swelling with new tissue, still gives the disc a "filled-in" appearance, and at this stage it is commonly white, or, more rarely, greyish in tint. As the new connective tissue contracts, it causes further narrowing of the vessels, which may be still partly covered over at their origin or in their course over the disc. Along their walls there are often lines of dead white tissue, due to thickening and degeneration in their outer coats, such as may often be seen bordering the long veins in the pia mater over the vertex of the brain. Ultimately, the contraction of the new tissue may cause a certain amount of excavation of the disc, and then, although the extremely white or grey colour of the disc indicates atrophy, there may be nothing to show that it is an atrophy following neuritis except the irregular margin of the choroid and the marked narrowing of the vessels. For a long time the disc remains white, ultimately it tends to become greyish, especially as seen by 'direct examination' and with a feeble illumination.

The diminution of vision during the period of subsidence, and the development of nerve atrophy, usually goes on increasing till the normal level of the disc is attained. After the stage of contraction has ceased, a slow improvement of vision may at times occur, especially in children or young people.

(β).—*Atrophy secondary to Embolism or Thrombosis of the Central Artery of the Retina*, is, in most cases, a well defined condition easily recognizable on account of the extreme narrowing of the arteries, which are reduced in size far below what is ever met with in neuritis. They pass over the disc and retina as fine red lines, or if, ultimately, it happens that any amount of collateral circulation is established, then the disused portion of the vessel remains as a narrow whitish line. The veins are at first a little dilated, but they soon become narrowed to a small extent. The retina around the macula lutea and also around the disc is usually more or less white and opaque, owing to the existence of œdema. The optic disc itself is white from the first and its edge is sharply defined, unless it happens to be dimmed by the œdema above referred to encroaching upon it. The whiteness goes on increasing till the disc presents just the same appearance that it does in simple atrophy, except for the extremely small size of the occluded vessels.

When one of the branches of the central artery happens to be occluded rather than the main trunk itself, then, in the province of such branch, and in this only, we meet with the characters above described.

But, whether the obstruction occurs in the trunk or in one of the main branches of the central artery, its occurrence is marked by the characteristic symptom of sudden and complete loss of sight in the one eye. In some cases the loss of sight has not been absolutely instantaneous, but has come on in the course of a few minutes. The loss of sight remains permanent where the obstruction is in the main vessel, and this is itself permanent; but where the obstruction is in one of the main branches only, the initial total loss of vision speedily clears up, so that it remains defective only in that portion of the field corresponding with the occluded branch.

Thrombosis of the ophthalmic artery (and, therefore, of the retinal artery also) occurs occasionally in association with thrombosis of the carotid and middle cerebral arteries. It would undoubtedly give rise in the fundus oculi to just the same appearances as are met with in embolism of the central artery. It is, however, apt not to be carefully observed, partly because of the gravity of the general state in such patients, and partly because some of the patients, in whom there is this association of tolerably sudden blindness of one eye setting in with a hemiplegia of the opposite side of the body, die in the course of a few days or weeks.

Again, cases are occasionally met with, in which there is reason to believe that thrombosis (rather than embolism) limited to the 'central artery' of the retina itself, is the cause of a more or less sudden blindness of one eye, leading to appearances otherwise similar. One such case has recently been under my observation, in which the patient (with no valvular disease of the heart, but with a history of syphilis) awoke one morning finding himself blind on the left side.

(γ).—*Atrophy following Choroido-retinitis.* The disc here may be either grey or white, as in simple atrophy, but according to Gowers it often presents special features, "being characterized by a peculiar reddish, or yellowish red tint of disc, uniform in distribution, the edges slightly blurred (or appearing so from the tint of the disc), and a marked wasting of the retinal vessels, which may be diminished in number as well as in size."

This choroiditic atrophy is said to be nearly always a consequence of syphilis, either inherited or acquired—especially of the former. It is, therefore, commonly regarded as one of the signs of inherited syphilis.

(δ).—*Atrophy following shocks to the Retina or blows upon the*

Eyeball. Failure in the nutrition of the retina, from any cause, is apt to entail, more or less quickly, an atrophy of the optic nerve, usually of the white variety. As Gowers points out, "now and then atrophy of the optic nerve follows a cause which seems to act by giving a shock to the retina, that leaves no trace behind, *e.g.*, the complete amaurosis, which may accompany the onset of embolism of one branch of the retinal artery, and is usually temporary, may sometimes be permanent, even though all the other branches of the retinal artery are pervious. Atrophy sometimes follows a blow on the eye, as in a case related by Laqueur, in which a blow caused complete amaurosis without visible change in the fundus, and simple atrophy followed. Such cases are of medical interest on account of the light they throw on the action of some general causes."

The *symptoms* caused by advancing atrophy of the optic disc are those of amblyopia, gradually increasing to complete amaurosis, such as have been already described.

C. THE THIRD, FOURTH, AND SIXTH CRANIAL NERVES.

These nerves supply all the muscles that move the eyeball, together with its intrinsic muscles, with the exception of the dilator iridis, which is innervated by the sympathetic.

Thus the **third nerve** supplies, in addition to the levator palpebræ superioris, the superior rectus, the internal rectus, the inferior rectus, the inferior oblique, together with the sphincter iridis and the ciliary muscle (or 'muscle of accommodation').

The **fourth nerve** supplies the superior oblique muscle.

The **sixth nerve** supplies the external rectus.

In reference to the movements of the eyeball, the principal points that need to be borne in mind are the directions in which the two oblique muscles tend to rotate the eye on its antero-posterior axis, viz., that the superior oblique rotates the eyeball from outwards, inwards and downwards, whilst the inferior oblique tends to rotate it outwards and upwards. This kind of movement of the eyeballs is required as a movement of adjustment, needful for correct vision (so that ocular pictures may fall upon proper parts of the retina), and in compensation for opposite movements of the head from shoulder to shoulder. If we place one hand upon each temporal region of a patient, and incline his head first to one shoulder and then to the other, we may see that the sound eye rotates in a direction opposite to that in which the head is lowered, while if either the superior or the inferior oblique muscle be paralysed in the other eye this rotatory movement is correspondingly lost or impaired therein.

It must, however, also be borne in mind that whilst the internal and the external recti suffice for movements of the eyeball directly inwards or directly outwards, the eyeball is elevated by the combined action of the rectus superior and oblique inferior, whilst it is depressed by the combined action of the rectus inferior and oblique superior. The conjoint action of the oblique muscles is needed in these cases, in order to correct certain slight rotations of the eye which would be caused by the single action of the rectus superior or inferior, owing to the fact that these latter muscles do not quite correspond with the vertical plane of the eyeball.

In consequence of what has just been said, it happens that in cases of double vision due to paralysis of either the internal or the external rectus the images are found to be parallel to one another (Fig. 95, 1, 2). On the contrary, in cases of paralysis of either of the other four muscles the affected globe becomes partially rotated on an oblique axis, owing

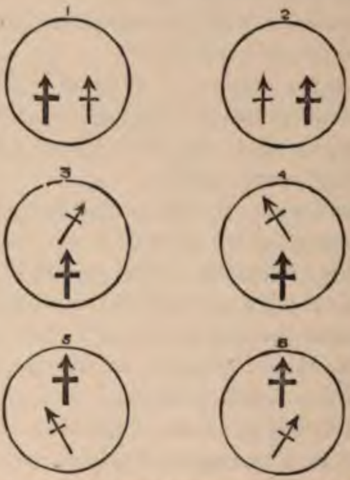

FIG 95.—RELATIVE POSITION OF THE DOUBLE IMAGES IN DIFFERENT FORMS OF DIPLOPIA [after Bristowe].

In the above diagram the thick cross represents the true image, the thin cross the false image. The left eye is supposed to be affected in each case.

1. With paralysis of internal rectus. 2. With paralysis of external rectus.
3. " " " superior rectus. 4. " " " inferior oblique.
5. " " " inferior rectus. 6. " " " superior oblique.

to the incompletely antagonised action of one or other of the oblique muscles; and the consequence is that the false image becomes tilted in a direction contrary to that of the rotation (Fig. 95, 3-6).

Before proceeding to speak of the distinctive effects resulting from paralysis of this or that ocular muscle, it will be needful briefly to consider certain **general symptoms**, or disturbances of vision, produced by such paralyses.

A paralysis of either of the muscles of the eyeball tends to show itself by some defect in the absolute mobility of the eye in the direction of the action of the paralysed muscle. This defective movement is only striking when the paralysis is complete. In simple paresis of the same muscle, the deficient movement is so slight that the defect is only detectable when the two eyes are carefully compared, in a manner subsequently to be referred to for the estimation of so-called 'secondary deviation.'

When in action, the paralytic or paretic muscle lags behind the healthy muscle of the opposite eye in concert with which it acts, in the extent of its contraction. The consequent limitation of movement becomes apparent to others as a squint or strabismus, while the immediate result to the patient himself is double vision, or diplopia.

In a case of paralysis of any orbital muscle, both the defective mobility and the diplopia increase, or become more obvious, when the eyes are directed towards the side of the paralysed muscle, and *vice versâ*. These characteristics are especially valuable in distinguishing paralytic from 'concomitant strabismus' (see p. 148).

The subjective result (diplopia) may present itself in its most troublesome or distressing form when the amount of ocular deviation (strabismus) is so slight as either not to reveal itself at all, or only after the most careful examination of the movements of the two eyes.

In the recognition of these slight forms of strabismus we must depend not so much upon the amount of primary deviation observable, but rather upon the amount of 'secondary deviation' that can be shown to exist.

Secondary deviation (that is, the deviation of the sound eye while the affected eye fixes) is greater than the primary deviation, because the muscle in the sound eye which is associated in its action with the paralysed muscle in the affected eye must receive a nervous impulse of equal intensity to that sent to the weak muscle. Hence the sound muscle becomes over-excited. The mode of testing this secondary deviation has been already described (p. 147).

Looking now to the subjective side of the defect, the diplopia, it will be found that the image formed on the retina of the affected eye is projected, or seems to lie, in the direction of the paralysed muscle. For instance, if the left external rectus be paralysed, the image of that eye will be formed to the inside (Fig. 96, *n*) of the macula lutea (*m*) and will, therefore, seem to lie to the left of the image belonging to the right eye.

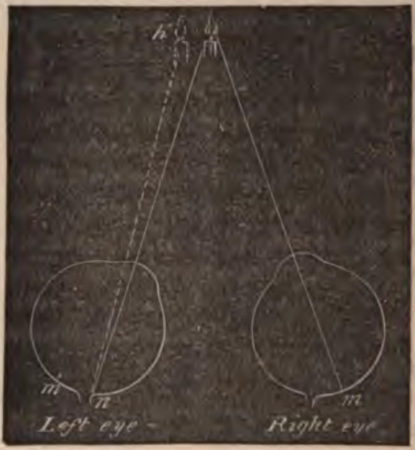

FIG 96. RELATIVE POSITION OF DOUBLE IMAGES IN PARALYSIS OF THE LEFT EXTERNAL RECTUS [after Swanzy].

Where the image of the affected eye lies to the corresponding side, as in this instance, we have what is termed homonymous diplopia, and such double vision always indicates convergence of the visual lines (internal squint).

But if the internal rectus of the left eye be paralysed the image on the retina of that eye falls to the outside of its macula lutea, and must therefore be projected to the right of the true position of the object, when we have what is called crossed diplopia, which must always be associated with divergence of the visual lines (external squint).

As the amount of ocular deviation varies in different cases, so does the amount of double vision or degree of separation of the images vary. The latter, however, may show itself to the patient when the former is incapable of being detected by the observer. In the minor amounts of diplopia there is mere haziness or indistinctness of vision, due to the overlapping of the two images to a varying extent with varying positions of the eye. In this state, vision may be perfectly good with each eye alone.

In all cases of diplopia the images may be best discriminated or referred to their respective eyes, by means of the colour test. A resort to this method is oftentimes absolutely needful in order to discriminate the true from the 'spectral image,' and consequently to enable us to decide upon the eye and the particular muscle that is at fault. The simplest means of applying the colour test is for the patient to look at some bright object, such as a lighted candle, placed at a distance of about ten feet directly opposite and on a level with his eyes, and for the observer to place before one of the eyes of the patient a piece of coloured glass. The latter will then be easily able to indicate to the observer the situation of the coloured image as well as of that belonging to the other eye. From the position of the two images relatively to the patient, even if he be not also guided by differences in their relative intensity, the observer will then be able to determine in which of the patient's eyes the true image is formed. The affected eye being thus made known, the situation of the spectral image—that is, whether it be to the right or to the left, above or below the true image—suffices to make known the particular muscle which is paralysed, either wholly or partially, if we bear in mind that the displaced image always appears on the side opposite to the ocular deviation, that is, towards the side on which the paralysed muscle exists.

It is important to remember that convergent strabismus is frequently associated with hypermetropia, and on rare occasions with myopia (p. 149). Both these forms of 'concomitant strabismus' are, however, generally unaccompanied by diplopia, owing to the fact that the patients so affected are soon enabled to avoid taking note of the displaced image. This is favoured by the image in the displaced eye being always formed on the same part of the retina, and by the amblyopia which soon ensues on the affected side.

A symptom that often occurs and causes much trouble in paralytic strabismus is a false projection of the visual field. A false judgment is formed concerning the position of external objects, owing to the fact that the amount of movement caused by the contraction of the affected muscle is apt to be over-estimated, since it is not in accord with the amount of energy expended. The field of vision is displaced in the direction of the action of the paralysed muscle—that is in the same direction as that in which the displaced image occurs. Thus, if a patient suffering from paralysis of one of the external recti grasps quickly at an object held before him, he is likely to miss it by going too far to the outer side. This false projection of the visual field, when the patient is walking or going up or down stairs, causes a feeling of great insecurity, together with distressing giddiness. Such effects are most marked when the paralysed eye is alone used, and are, therefore, not to be confounded with any discomfort arising from the mere perception of double images.

These troublesome symptoms, of both orders, may be obviated by covering the affected eye so as altogether to prevent its use. Short of this, however, the patient soon learns to hold his head in a peculiar position in order that images may fall on those parts of his retinæ which do not give rise to double vision. These positions of the head differ in accordance with the different muscles that are paralysed, and thus come to be in themselves characteristic.

After a time, secondary contracture is apt to occur in the unantagonized muscle of the affected eye, owing to loss of the opposition formerly exercised by the now paralysed muscle. It is obvious that the effect of this contracture must be to increase the amount of pre-existing diplopia, that is, to widen the distance between the double images.

THE THIRD NERVE (OCULO-MOTORIUS).

The third is an exclusively motor nerve which supplies different muscles of, or in connection with, the eyeball. Its fibres have their deep origin in an elongated nucleus lying partly beneath the aqueduct of Sylvius and partly on the anterior part of the floor of the fourth ventricle (Fig. 97). They reach the surface of the brain at the posterior and inner part of the cerebral peduncle. Here the nerves of opposite sides diverge from one another (Fig. 87) crossing the optic tracts almost at right angles in order to pass through the dura mater and gain the orbital cavity. Within the orbit each nerve supplies the following muscles :—the levator palpebræ superioris ; the superior, inferior, and internal recti ; the sphincter of the iris ; and the ciliary

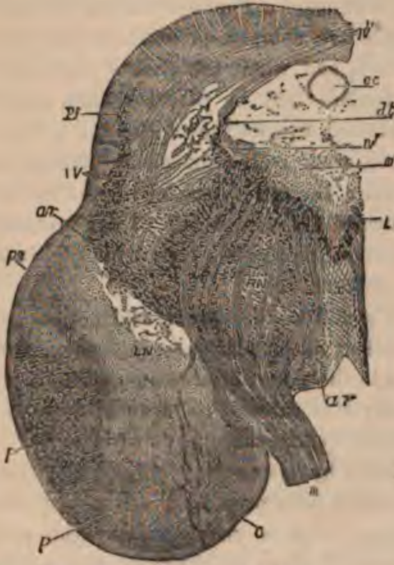

FIG. 97. TRANSVERSE SECTION OF THE CRUS CEREBRI, ON A LEVEL WITH THE ANTERIOR PAIR OF CORPORA QUADRIGEMINA AND THE ROOTS OF THE THIRD NERVE, FROM A NINE-MONTHS EMBRYO [after Krause and Ross].

C, C, Crusta. P, Pyramidal tract. *p*, accessory portion of the pyramidal tract. LN, Locus Niger. RN, Red nucleus of the tegmentum. L, Posterior longitudinal fasciculus. *ar* and *ar′*, upward continuation of the internal and external portions respectively of the anterior root-zone of the spinal cord.

III, Third nerve; III′, nucleus of the third nerve.

IV, Fourth nerve; IV′, nucleus of the fourth nerve. IV″, Crossing of the fibres of the fourth nerve to opposite sides.

dt, Descending root of the trigeminus. *cc*, Aqueduct of Sylvius. *x*, Crossing of the fibres of the superior peduncles of the Cerebellum. *pf*, Fasciculus of medullated fibres proceeding to the anterior pair of Corpora Quadrigemina.

muscle. The different parts of the nucleus which innervate these several muscles have been thought to be determined by some observers, but their respective situations cannot as yet be said to be definitely settled.

In some cases the paralysis of the nerve is complete; that is, all its fibres are implicated. In other cases the fibres supplying individual muscles may be affected, so that weakness or actual paralysis may exist in this or that muscle only.

Complete paralysis of the third nerve is to be recognized by the following signs:—The upper eyelid is dropped and cannot be raised; that is, there is complete ptosis. When it is raised by the observer, the eyeball is found to be in its natural position or turned slightly outwards and downwards. The movements inwards, upwards and directly downwards are completely lost; on the other hand, it can be moved outwards, and when the patient is asked to move the eye downwards the eyeball may be seen to rotate most distinctly in a downward and outward direction, owing to the action of the superior oblique and the external rectus muscles. Where the paralysis has existed for some time, the eye is generally found to be turned decidedly outwards and downwards as a result of the unbalanced action and 'secondary contraction' of the two sound muscles, the external rectus and the superior oblique.

The pupil is in a mid state between contraction and dilatation, and it does not contract at all on exposure to light. Owing to paralysis of the ciliary muscle the power of accommodation is also lost. Again, owing to the paralysis of three of its straight muscles, the eyeball may be slightly protruded (exophthalmus paralyticus).

Fixation of objects by both eyes is only possible in a downward and outward direction, and false projection of the field of vision occurs in all other directions; double vision is also the result in all other directions, producing distress and vertiginous sensations, so that even if there be not complete ptosis the patient commonly prefers to keep the affected eye closed, and use the other only.

The nerve fibres composing some one branch of the third nerve, or those supplying some two of the muscles above named may be affected separately; and thus give rise to various partial paralyses in the domain of this nerve.

When the **branch supplying the levator palpebræ superioris** is affected, more or less complete *ptosis* is produced. The eyelid drops over the ball, so as more or less completely to obliterate the palpebral fissure. The paralysis of this muscle is, in some cases, associated with that of the superior rectus, since both these muscles are supplied by the same branch of the third nerve. It is supposed that ptosis very often has a peripheral origin; that is, may be caused by injury or disease of the nerve filament supplying this particular muscle.

It has been thought, however, that some of the cases in which paralysis of the levator palpebræ superioris occurs alone may be due to small lesions in a definite region of the cortex. On this subject Ferrier says:—"Some such cases have been observed in connection with disease of the cortex, and attempts have been made, but not I think successfully or in accordance with experimental lesions, to localise this centre in the angular gyrus. If a distinct volitional centre for the levator palpebræ superioris exists in the human brain I think it must be sought for in the region now under consideration "—that is, somewhere in the region marked (12) in Fig. 6. It is Grasset and Landouzy especially who believe that clinico-pathological evidence points rather in the direction of the angular gyrus. This difference of opinion has already been referred to (p. 96), as part of a larger question, concerning the region of the cortex principally concerned with the production of 'conjugate deviation of the eyes and head.' Ferrier calls attention to a fact which I have also observed, that in very many persons one lid droops more than the other over the cornea (generally the left), and that this drooping may be greatly intensified in low states of the system or from over fatigue.

Either one of the three recti muscles supplied by the third nerve may also at times be paralysed singly; and in each case double vision, as well as some amount of alteration in the position of the eyeball, results. The inferior oblique is rarely paralysed alone.

The signs and symptoms indicative of paralysis of these four muscles supplied by the third nerve as well as of the two other ocular muscles supplied by the fourth and sixth cranial nerves (superior oblique and external rectus), will be best understood if we arrange them in a tabular form, so as to bring out their mutual contrasts, and thus facilitate diagnosis in any doubtful case.

Signs and Symptoms.	Internal Rectus.	External Rectus.	Inferior Oblique.
Appearance and Movements of Affected Eye.	Deviation of eye outwards. Movements inwards defective, often not beyond median line.	Deviation of eye inwards. Movements outwards defective, often not beyond middle line.	Slight deviation downwards and inwards. Slight defective mobility, most marked in direction upwards and inwards.
Secondary Deviation of Sound Eye.	Outwards.	Inwards, and well marked.	Upwards and somewhat outwards.
Relative position of Double Images.	Side by side, parallel, and crossed—the false image being on the healthy side. Separation of images increasing with movements of object towards sound side. Diminution of distance and ultimately single vision with movements of object towards affected side.	Side by side, parallel and homonymous — the false image being on the affected side. Separation of images increasing with movements of eye outwards. Diminution and ultimately single vision with movements inwards.	One above the other; the false image above and tilted towards the affected side. Vertical distance of images increases as object is moved inwards; and inclination to one another increases with rotation outwards. Single vision with eyes directed downwards.
False Projection of Visual Field.	Inwards.	Outwards.	Upwards and somewhat outwards—on fixation in the median plane.
Vertigo.		Often well marked.	
Attitude assumed for Mitigation of Double Vision.	Head turned towards healthy side.	Head turned towards the affected side.	Head thrown backwards and chin turned slightly towards the healthy side (so as to bring the lower and outer segment of the field of vision chiefly into use).
Effects of Secondary Contracture of Antagonist Muscle.	Double images extend into the outer half of the visual field.	Double images extend inwards, and may affect the whole field of vision.	Double images extend into lower half of the visual field.
Association with other Paralyses.	Rare as an isolated affection; commonly as part of a general paralysis of the third nerve.	Separately, or in association with that of superior oblique. More rarely bilateral.	Rare as an isolated affection; and when coexisting with paralysis of other muscles supplied by the third nerve its distinct diagnosis becomes impossible—ptosis masking many of above signs.

Signs and Symptoms.	Superior Rectus.	Inferior Rectus.	Superior Oblique.
Appearance and Movements of Affected Eye.	Deviation slightly downwards; cornea diverges a little outwards from action of inferior oblique. Upward movements defective.	Deviation slightly upwards; and cornea turned a little outwards owing to action of superior oblique. Downward movements defective.	Slight deviation of eye inwards and upwards on lowering the object; and simply upwards when it is moved far towards the healthy side. Slight defective mobility, chiefly downwards and inwards.
Secondary deviation of Sound Eye.	Upwards and somewhat outwards.	Downwards and somewhat outwards.	Straight downwards, and generally well marked.
Relative position of Double Images.	In upper half of visual field; images one above the other; the false image above and tilted towards sound side. Separation increasing with elevation of point of fixation; and diminishing to single vision with depression of visual axis to horizontal line.	In lower half of visual field; images one above the other; false image below and tilted towards the affected side. Separation increases as axis of vision is directed more downwards; distance diminishing to single vision with elevation of visual axis.	In lower half of visual field; one below the other; false image below and tilted away from affected side. Vertical distance greatest when looking downwards and inwards. Inclination greatest when looking downwards and outwards. (Image of affected eye seems nearer than that of sound eye.) Single vision with eyes directed upwards.
False Projection of Visual Field.	Upwards.	Downwards.	Downwards and slightly outwards.
Vertigo.	Rare; felt principally on ascending ladder or stairs.	Very marked in all cases where line of vision has to be lowered as when walking, ascending stairs, and during all kinds of handiwork.	Often well marked.
Attitude assumed for Mitigation of Double Vision.	Head and field of vision depressed.	Head inclined forwards.	Head inclined forwards and turned towards healthy side (the affected eye being rotated towards the chin), so that objects are brought into the upper and outer part of the field of vision.
Effects of Secondary Contracture of Antagonist Muscle.	Double images extend into the lower half of the field of vision.	Double images extend into the upper half of the field of vision.	Double images extend into the upper half of the field of vision.
Association with other Paralyses.	Sometimes associated with ptosis, or may exist alone.	Mostly as part of a general paralysis of the 3rd nerve.	Separately, or in conjunction with the external rectus.

If several of the ocular muscles, as not unfrequently occurs, are coincidently paralysed, the symptoms are combined and may become very complicated and difficult to decipher.

Paralysis of the Sphincter iridis (Mydriasis paralytica).—In this affection the pupil is in a medium state of dilatation, and is nearly or completely motionless when subjected to the stimulus of light; it mostly contracts very little, or not at all, when the eyes are strongly converged, or when efforts of accommodation are made. The pupil may be still further dilated by atropine. Vision is rendered indistinct in consequence of circles of dispersion; and bright light is intensely disagreeable. The power of accommodation, though often diminished, may be completely preserved.

Paralysis of the Musculus accommodatorius (Ciliary muscle).—This affection may occur quite independently, but is often also complicated with mydriasis. In this form of paralysis the focal distance is increased; the patient finds himself incapable of focussing near objects, or of reading small print. He often forms an incorrect estimate of the size and distance of objects. Paralysis of accommodation is one of the most common symptoms of diphtheritic paralysis; it may also accompany any other paralysis of the third nerve.

THE FOURTH NERVE.

The fourth nerve supplies the superior oblique muscle only. The signs and symptoms attendant upon the paralysis of this muscle have already been given in tabular form (p. 423).

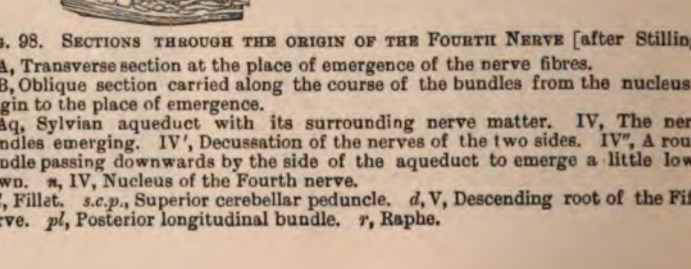

FIG. 98. SECTIONS THROUGH THE ORIGIN OF THE FOURTH NERVE [after Stilling].

A, Transverse section at the place of emergence of the nerve fibres.

B, Oblique section carried along the course of the bundles from the nucleus of origin to the place of emergence.

Aq, Sylvian aqueduct with its surrounding nerve matter. IV, The nerve bundles emerging. IV′, Decussation of the nerves of the two sides. IV″, A round bundle passing downwards by the side of the aqueduct to emerge a little lower down. *n*, IV, Nucleus of the Fourth nerve.

l, Fillet. *s.c.p.*, Superior cerebellar peduncle. *d*, V, Descending root of the Fifth nerve. *pl*, Posterior longitudinal bundle. *r*, Raphe.

The origin of these nerves is from a nucleus which lies in the region between the anterior and the posterior quadrigeminal bodies, immediately below that of the third nerve as shown in Fig. 98. The two nerves subsequently decussate in the substance of the valve of Vieussens. Their relations at the base of the brain, at their exit from the skull, and within the orbit are shown in Figs. 87, 88, 100. Throughout this long course the fourth nerves may be affected in various modes, and in all cases the recognition of the seat and nature of the lesion in which it is involved will depend, in part, upon a study of the associated morbid signs, whose nature is apt to change in accordance with its varying anatomical relations in different parts of its course from its nucleus of origin to the superior oblique muscle.

The fact of the two nerves decussating in the substance of the valve of Vieussens, makes it possible to have a double paralysis of these nerves as one of the signs of a tumour of the middle lobe of the cerebellum. The fact that this is a very rare event in such cases is, doubtless, to be ascribed to the absence of a resisting surface on the other side of the valve of Vieussens—that is, on the side of the fourth ventricle.

The questions relating to the pathological diagnosis will, however, be dealt with further on.

THE SIXTH NERVE.

The sixth nerve supplies one muscle only, viz., the external rectus. The signs and symptoms to which its paralysis gives rise have been already set forth in a tabular form (p. 422).

The sixth nerves arise from nuclei situated pretty close together on each side of the middle line, slightly below the surface of the fourth ventricle (Fig. 99). From these points the nerve fibres pass downwards and forwards so as to emerge in the groove between the pons and the medulla. It is especially worthy of note that the fibres of the facial nerve wind round and come into the closest relations with the nucleus of the corresponding sixth nerve (Fig. 75). Hence a small focal lesion on one side of the medulla may, among other signs, give rise to a simultaneous paralysis of muscles supplied by the sixth and the seventh nerves—a combination which is of importance from the point of view of regional diagnosis.

The relations of the sixth nerves at the base of the brain, at their exit from the skull, and within the orbit, may be gathered from a reference to Figs. 87, 88, 100.

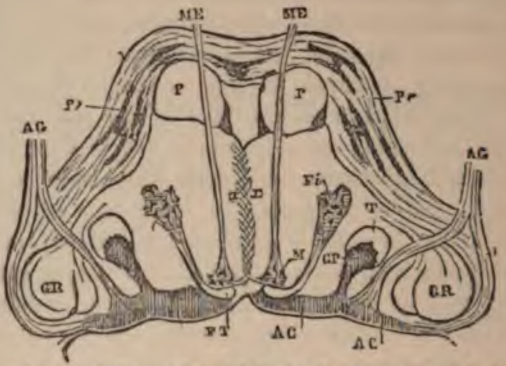

FIG. 99. DIAGRAM OF A SECTION AT THE LINE OF JUNCTION BETWEEN THE BULB AND THE PONS [after M. Duval].

P,P, Anterior pyramids. Pr,Pr, Transverse fibres of the pons between the layers of which masses of grey matter are irregularly disposed.

ME,ME, Roots of the nerves supplying the external recti (6th nerves). M, nucleus of the sixth nerve, with which the root fibres of the facial nerve come into close relation (see Fig. 80, RVII).

FT, fasciculus teres (portion of the loop of the facial).

Fi, Inferior nucleus of the facial (in which the fasciculus teres takes its origin).

GP, Head of the posterior cornu. T, Ascending root of the trigeminus.

AC, Grey substance of the floor of the 4th ventricle (nucleus of the auditory).

AG, Root fibres of the auditory nerve.

GR, GR, Restiform bodies.

The questions relating to the *pathological diagnosis*, in cases of paralysis of muscles supplied by this, as well as by other ocular nerves, will be dealt with further on, in a separate section.

Spasms of the Ocular Muscles.

Spasms, both tonic and clonic, may occur from time to time in the extrinsic as well as in the intrinsic muscles of the eyeball. Those of the **tonic order** are by far of the most importance, and as they are often produced by a minor action of just such causes as, in a more marked form, are apt to produce paralyses, some brief reference is needful to them here.

Slight pressure may have an irritative effect productive of spasm, where a more severe pressure would lead to paralysis. Although the difference in the cause is slight, the effects produced may thus be diametrically opposite—since *the spasm of any given ocular muscle produces just the same kind of deviation of the eye as would be induced by a paralysis of its antagonist.*

The *levator palpebræ superioris* is only very rarely the seat of tonic spasm. The effect, when it exists, is to cause the upper lid to be permanently raised, so that the eye is not closed even during sleep. This rare condition may be induced by centric irritation, though it is much more likely to be caused by some peripheral irritation acting within the orbit upon the nerve which supplies this muscle, or even to be produced in a reflex fashion by some peripheral irritation implicating one or other of the branches of the fifth nerve.

Of the muscles that move the eye ball, the *internal rectus* is by far the most frequently affected by spasm. It may be caused by incipient cerebral disease or meningitis; or it may be induced by some reflex irritation, especially in children. It is often associated with neuralgic pains on the same side of the head.

These primary spasms of the internal rectus must not be confounded with the secondary spasm of the same muscle that is apt to occur when its antagonist, the external rectus, has been paralysed for a long time. Spasm of the internal rectus gives rise to an homonymous diplopia, with parallel arrangement of the images, exactly resembling that which occurs when the external rectus is paralysed (Fig. 95, 2). There is, however, one important peculiarity serving to distinguish the two cases. In paralysis of the external rectus the images are steady and fixed in their relative positions; in spasm of the internal rectus, on the other hand, the two images are in constant oscillation, alternately approaching and receding from one another.

But in the majority of cases in which a spasmodic internal squint is met with, it is due neither to disease of the brain nor to irritation of the branch of the third nerve; it is caused rather by defects in the eyeball leading to prolonged over-action of the internal recti, *e.g.*, hypermetropia and myopia (see p. 148).

A spasmodic external squint caused by *primary tonic spasm of the external rectus* is met with only with extreme rarity. It has been found in locomotor ataxy alternating with spasm of the internal rectus. Spasm of the external rectus gives rise to a diplopia of the same kind as that met with in paralysis of the internal rectus (Fig. 95, 1), except that in the latter the two images are fixed, and in the former they oscillate.

Secondary spasm of the external rectus occurs more frequently, in cases where its antagonist, the internal rectus, has been long paralysed.

Apart from the causes already referred to primary spasms of the internal and the external recti, but especially the former, are apt to be produced by the same kind of conditions, direct and reflex, as were mentioned in reference to spasm of the levator palpebræ superioris.

Nothing positive seems to be known concerning primary spasms of the superior and inferior recti, or of the upper and lower oblique muscles.

Peculiar **clonic spasms** are apt to involve the muscles of the eyeballs, giving rise to the condition known as nystagmus, in which there recur from time to time remarkable oscillatory or rotatory movements of the eyeballs. The movements are simply lateral and oscillatory when the external and the internal recti are principally concerned; on the other hand, they assume more of a rotatory character when the oblique muscles are implicated. Lateral nystagmus may be frequently seen in healthy individuals, especially in females, when they look at a quick succession of passing objects, as when two trains are passing one another and the person in the opposite corner seat looks at the advancing carriages as they pass. When it occurs as a morbid sign, also, nystagmus is generally much more marked on regarding somewhat distant objects, than when the eyes are fixed upon some near point.

Nystagmus occurs with special frequency in cases of disseminated or cerebro spinal sclerosis, and some suppose it to be due in these cases to the occurrence of patches of sclerosis in the cerebellar peduncles or in the cerebellum itself. It occurs also, though more fitfully, in connection with other diseases of the brain, especially with chronic hydrocephalus. Again, it is apt to be met with in association with certain congenital defects in the eyes; it occurs, for instance, not unfrequently in albinos.

Colliers who work in constrained positions, and consequently have to make a somewhat unnatural use of their eyes, are not unfrequently subject to so-called 'miners'' nystagmus ('Brain,' Vol. III, p. 173).

Nystagmus may also, at times, occur as a temporary condition, resulting from some peripheral irritation.

Tonic spasm of the sphincter of the iris has already been referred to as a sign produced by different conditions, and consequently occurring with comparative frequency. It may be produced by irritation of the third nerve in any part of its course, or by centric irritation of its nucleus. It is frequently associated with irritation of the branch of the third nerve that supplies the internal rectus. It may be produced by the action of various drugs. When contraction of the pupil is due to paresis or paralysis of the sympathetic nerves supplying the iris, the loss of power in the dilator fibres causes the pupil no longer to respond so freely, if it should respond at all, to the action of atropine.

Tonic contraction of the ciliary muscle is very apt to be associated with tonic contraction of the pupil, to which we have just been referring. They are commonly associated muscular actions, and are, therefore, apt to be excited simultaneously by morbid irritations. Consequently, no distinct reference need be made to this condition.

Clonic spasms of the sphincter of the iris occur only rarely. In this condition contractions and relaxations of the pupil quickly alternate with one another. It is known by the name of hippus, or 'chorea of the iris.' A minor phase of this condition is extremely common in epileptic patients.

The Pathological Diagnosis in Paralyses of Different Ocular Muscles.

The problems of diagnosis in regard to paralysis of this or that ocular muscle are very closely interwoven with the questions concerning the etiology of such paralyses—or, in other words, the regional and the pathological diagnosis are closely related questions, which often shed a mutual light upon one another.

The nerves going to the ocular muscles may be affected in one or other of three principal situations; either (*a*) within the orbit, (*b*) within the cranium at some point between their passage through the dura mater and that part of the surface of the brain with which they are connected, or (*c*) by implication of their root fibres or nuclei within the base of the brain.

Concerning the intra-cerebral course of the fibres of these nerves towards the cortex almost nothing is known; and unilateral lesions above the level of their respective nerve nuclei would seem to give rise to no corresponding paralysis, though a possible exception to this may be made as regards the branch of the third nerve that supplies the levator palpebræ superioris, and the paralysis of which gives rise to ptosis (see p. 420).

The paralyses of these nerves of the eyeball are, therefore, all of them of the so-called peripheral type; for, as we have already pointed out (p. 11), lesions of the nuclei of origin of nerves give rise to paralyses which, though they are often classed as central, exhibit all the typical characters of well-marked peripheral paralyses.

(*a*).—One of the most frequent of all the varieties of paralysis of the ocular muscles is the so-called *rheumatic paralysis*, probably a form of neuritis, caused principally, as commonly supposed, by exposure to cold. The sixth and one or more branches of the third nerve are those chiefly affected by this cause; the fourth nerve only very rarely.

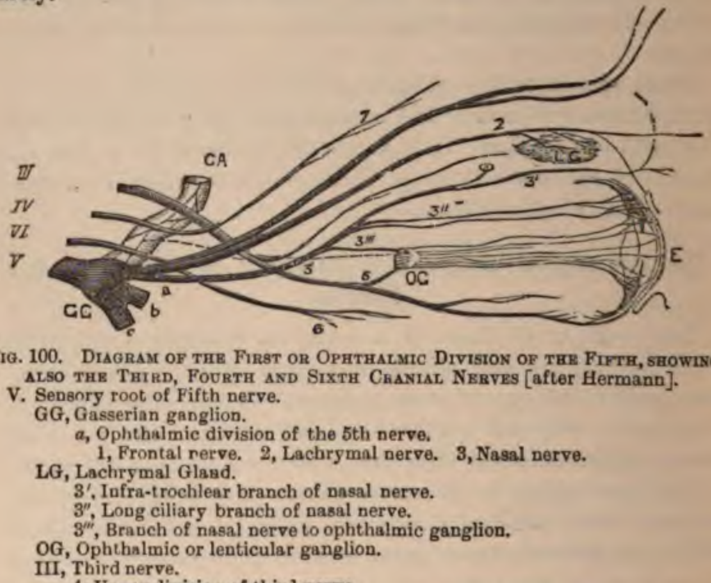

FIG. 100. DIAGRAM OF THE FIRST OR OPHTHALMIC DIVISION OF THE FIFTH, SHOWING ALSO THE THIRD, FOURTH AND SIXTH CRANIAL NERVES [after Hermann].

V. Sensory root of Fifth nerve.
  GG, Gasserian ganglion.
    a, Ophthalmic division of the 5th nerve.
      1, Frontal nerve. 2, Lachrymal nerve. 3, Nasal nerve.
  LG, Lachrymal Gland.
    3′, Infra-trochlear branch of nasal nerve.
    3″, Long ciliary branch of nasal nerve.
    3‴, Branch of nasal nerve to ophthalmic ganglion.
  OG, Ophthalmic or lenticular ganglion.
  III, Third nerve.
    4, Upper division of third nerve.
    5, Lower " " " ", near point where it gives the short root to the ophthalmic ganglion.
  IV. Fourth nerve.
    7, Its fibres passing to the superior oblique muscle.
  VI. Sixth nerve.
    6, Its fibres passing to the external rectus muscle.
  CA, Carotid Artery.
  E, Vertical section through anterior part of eyeball; conjunctiva indicated by dotted line.

Penetrating wounds of the orbit, produced in the most various ways, are also very frequent causes of paralysis of one or other of the ocular muscles. Again, one or other of the nerves within the orbit may be involved or pressed upon by orbital tumours, extravasations of blood, exostoses, etc. The nerves in this situation may also be affected by an inflammatory process, as when an erysipelatous inflammation from the outside invades the tissues of the orbit, or when a basal meningitis extends outwards.

Ophthalmoplegia interna is the name that has been given by J. Hutchinson to indicate the combined paralysis of all the muscles within the eyeball, viz., both the circular fibres and the dilators of the iris, as well as the ciliary muscle. This condition Hutchinson believes to be due to disease of the lenticular ganglion, for the most part induced by syphilis.

In this connection it may be well to state that paralysis of the radiating fibres of the iris alone may occur (paralytic myosis), just as paralysis of the sphincter of the iris may occur alone (paralytic mydriasis). In paralytic myosis the pupil is extremely contracted and dilatation cannot be brought about by atropine. It may be caused by disease of the sympathetic root of the lenticular ganglion, by disease of the cervical trunk of the sympathetic, or, lastly, by injury or disease occurring in the ciliospinal region of the cord.

(*b*).—Within the cranial cavity the motor nerves of the eye may be affected in the most various modes (Fig. 88); they may be pressed upon or involved by tumours originating in the brain, the meninges, or the bone; they may be pressed upon by aneurysms of the carotid artery; they may be involved in thickenings of the dura mater, or in localised chronic inflammations of this membrane; or, they may be compressed against the bone by hæmorrhages occurring into the meninges.

(*c*).—Diseases of the brain itself may cause paralysis of the motor nerves of the eye in various modes. This occurs with especial frequency in the case of tumours involving the cerebral peduncles or the pons. Paralysis of the third nerve (Fig. 96) in association with peduncular disease produces an altogether typical grouping of symptoms (p. 325). The sixth nerves are especially apt to be involved by disease occupying the lower part of the pons, or by any morbid process implicating their nuclei beneath the floor of the fourth ventricle (Fig. 99). Degenerative disease affecting the nuclei of the third and fourth nerves in their situation beneath the Sylvian aqueduct will also have to be referred to presently, as the cause of a peculiar combination of paralyses of the ocular muscles, known as ophthalmoplegia externa. Again, tumours or hæmorrhages situated in the crura cerebri may compress the third or fourth nerves to a serious extent, and so also may dropsical distensions of the third ventricle.

Syphilis is a very frequent cause of paralysis of the ocular muscles. One or other of the motor nerves of the eye, and the sixth with especial frequency, may become affected in various ways; they may, for instance, be involved in gummatous thickenings at their origin or in some part of their course, and also by inflammatory thickenings of the dura mater at their points of exit from the skull. They may, likewise, be implicated by periostitis or extoses within the orbit, occurring as late manifestations of syphilitic infection.

Ophthalmoplegia externa is a disease of progressive onset in which all the extrinsic ocular muscles of both sides tend to become more and more paralysed. It shows itself first by drooping of the eyelids; then by increasing paresis of the other ocular muscles till at last the power of moving the globes becomes more or less lost in all directions. Hutchinson has found that in one-third of the recorded cases, there has also been blindness with white atrophy of the optic nerves. More rarely other cranial nerves become implicated.

It has long been recognized that this paralysis is of a progressive type, and it has been assumed to be due to degenerative pathological conditions similar to those causing 'progressive muscular atrophy,' and 'glosso-labio-laryngeal paralysis.' This expectation was realized by Gowers, who found, in one of Hutchinson's cases, atrophy and disappearance of the nerve cells in the medullary nuclei of the third, fourth and sixth nerves, together with corresponding changes in their root fibres.

This condition does not seem to associate itself specially with similar changes occurring in the spinal cord (progressive muscular atrophy), though it has been met with by Hutchinson and others as an accompaniment of locomotor ataxy.

During convalescence from some acute diseases paralysis of one or more ocular muscles occasionally occurs. This is especially common during or after *Diphtheria*, when certain branches of the third nerve are prone to be affected—leading especially to dilatation of the pupil and paralysis of accommodation.

The only *disease of the Spinal Cord* in which we are entitled to look for the frequent association of paralysis of any of the ocular muscles is locomotor ataxy. In its early stages paresis of the sixth or of some branches of the third nerve are especially apt to show themselves, revealing their presence principally by causing temporary diplopia and it may be vertigo. Ophthalmoplegia externa may also be met with, as above indicated.

What has been said above in regard to etiology, is of such a nature as to render it needful to give only a few further hints in regard to **the general mode of arriving at a diagnosis,** in cases of paralysis or of spasm of ocular muscles.

The particular ocular muscle or muscles affected with paralysis or spasm has, in each case, to be decided altogether by reference to the presence or absence of such signs as have been already specified (pp. 422, 423).

When the localization of the paralysis or spasm has been decided upon (as involving this or that muscle), we then have to consider whether such paralysis or spasm is likely to be due to central or peripheral causes. It is well, if possible, to exclude the former, before attempting to decide between the different peripheral causes, one or other of which may be in action in the particular case before us.

As far as concerns diseases of the *Spinal Cord,* the existence or not of locomotor ataxy must be considered in reference to diplopia, or to contraction of the pupil with loss of its reflex to light and presence of contractility during accommodation (Argyll-Robertson phenomenon); and, again, it must be borne in mind that spasmodic mydriasis or paralytic myosis may be caused by irritative or destructive disease in the cilio-spinal region of the spinal cord.

In deciding that paralyses or spasms of the ocular muscles are due to *Intra-cranial Disease,* we must be supported by the existence of other good evidence in favour of such disease—we must look, that is, for the coexistence of partial or complete hemiplegia, or for simultaneous paralyses of other cranial nerves, the presence of which would lend support to this notion. General evidence supporting the existence of an intra-cranial new growth, or of meningitis, either acute or chronic, must also be taken into account. Where the conclusion is arrived at that the cause of certain ocular symptoms is to be attributed to brain disease, the probable locality of such disease has, of course, to be considered in reference to the sum total of signs and symptoms present. In this relation, it is altogether a subordinate question whether the intra-cerebral origins of the several nerves are affected, or whether the nerves themselves are pressed upon or involved by some morbid product situated anywhere within the cranium—though these are points that must, of course, subsequently receive our full consideration.

Although such paralyses as have been referred to in the last paragraph are, as we have already indicated (p. 430), truly of a 'peripheral type,' still it is most convenient to regard, as *peripheral affections*, only those cases where the nerves are involved outside the cranial cavity. Having excluded the likelihood of intra-cranial lesions, the existence of this or that cause already referred to as possibly operating within the orbit (p. 430) must be considered. In this class of cases, it is well to bear in mind that isolated paralyses of ocular muscles are often complete; while this is rarely so when they are of centric origin.

The squint with possible diplopia, that may be caused by hypermetropia and myopia, must not be confounded with cases in which such symptoms are caused by lesions of nerves supplying the ocular muscles (see p. 148).

D.—THE FIFTH OR TRIGEMINAL NERVE.

This nerve contains a large sensory and a small motor portion. Reference to Figs. 101, 105 (v, v', v",) will clearly show the positions

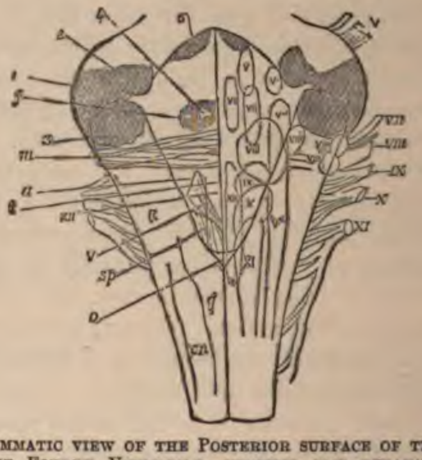

FIG. 101. DIAGRAMMATIC VIEW OF THE POSTERIOR SURFACE OF THE MEDULLA, OR FLOOR OF THE FOURTH VENTRICLE, SHOWING THE RELATIVE POSITIONS OF DIFFERENT NERVE NUCLEI [after Erb].

The left half of the figure (represents:—*Cn*, funiculus cuneatus, and *g*, funiculus gracilis; O, obex; *sp*, nucleus of the spinal accessory; *p*, nucleus of the pneumogastric; R, restiform body; XII', nucleus of the hypoglossal; *t*, funiculus teres; *a*, nucleus of the acousticus; *m*, striæ medullares; 1, 2, 3, middle, superior and inferior cerebellar peduncles. *f*, fovea anterior; 4, eminentia teres (genu nervi facialis); 5, locus cœruleus.

The right half of the figure represents the nerve nuclei diagrammatically:—V, motor trigeminal nucleus; V', median, and V", inferior sensory trigeminal nuclei; VI, nucleus of abducens; VII, facial nucleus; VIII, posterior median acoustic nucleus; VIII', anterior median, VIII", posterior lateral, VIII''', anterior lateral acoustic nuclei; IX, glosso-pharyngeal nucleus; X, XI, and XII, nuclei of vagus, spinal accessory, and hypoglossal nerves respectively. The Roman numerals at the side of the figure from V to XI represent the corresponding nerve roots.

in the pons and medulla of the sensory and motor nuclei of this nerve, and their relations with other nerve nuclei. The sensory and motor fibres in connection with these nuclei, appear, as is well known, at the surface near the middle of each lateral region of the pons. In the natural position of the body the small motor portion lies underneath the sensory division, passing beneath the Gasserian ganglion (Fig. 88), it then immediately joins the third or infra-maxillary branch of this division of the fifth. The other two branches—viz., the ophthalmic and the superior maxillary—are, therefore, purely sensory nerves.

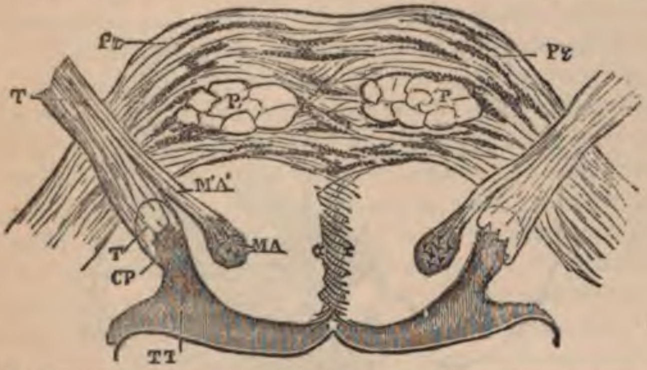

FIG. 102. DIAGRAM OF A SECTION THROUGH THE PONS AT THE LEVEL OF THE EMERGENCE OF THE FIFTH NERVES [after M. Duval].

P,P, Pyramidal strands. Pr, Transverse fibres of the pons with interstratified masses of grey matter. TT, Grey substance of the floor of the fourth ventricle (locus cœruleus). CP, Gelatinous substance of Rolando. T, Ascending root of the trigeminus, curving so as to emerge as part of the larger root of the trigeminus. MA, Motor nucleus of the trigeminus. M'A', Motor root of the trigeminus, (masticatory nerve). T', The Fifth Nerve at its point of emergence from the pons.

It is important to bear in mind that the third, the fourth, and the sixth nerves are, at the base of the brain, close to one another and to the fifth nerve ; and, moreover, that they pass through the dura mater at a very small distance from one another (Figs. 88 and 99).

Lesions of the fifth nerve are apt to produce varied effects, partly sensory, partly motor, while they may also entail certain disorders of secretion and of nutrition. Secretion is affected, because the nerve contains fibres which proceed to the lachrymal, parotid and submaxillary glands. Nutrition is impaired for a reason which is not as yet beyond the reach of doubt. It is very commonly supposed that the nerve contains certain special trophic fibres which have their origin in the Gasserian ganglion ; and that the injury of these fibres accounts for the occurrence of inflammations of the eyeball, ulcerations of the cornea, and also ulcerations of the mucous membrane of the mouth—effects that are undoubtedly apt to occur in association with some diseases of the fifth nerves. It seems possible, however, that these effects may be produced by means of irritations transmitted to the tissues in a centrifugal direction, through the ordinary sensory fibres of the nerves—that they may, in fact, be effects occurring quite independently of the existence of any special trophic fibres. Vaso-motor fibres for the conjunctiva and the iris are also distributed with the trigeminus.

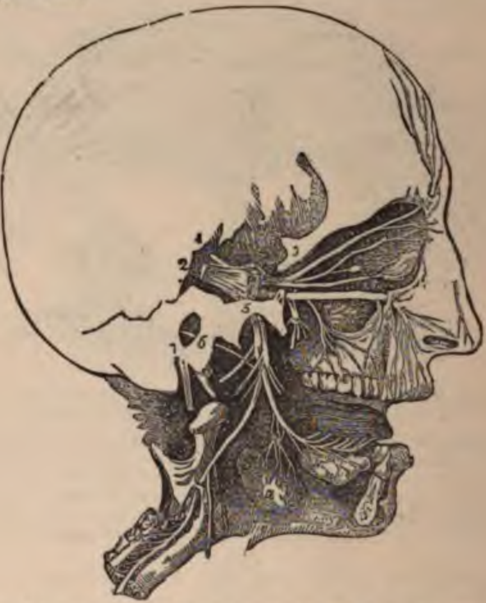

FIG. 103. GENERAL PLAN OF THE BRANCHES OF THE FIFTH PAIR FROM QUAIN'S 'ANATOMY' [after sketch by Sir Chas. Bell].

1, small root of the Fifth Nerve.
2, Large root passing into Gasserian ganglion.
3, Placed on bone above ophthalmic nerve, which is dividing into frontal, lachrymal, and nasal branches, the latter connected with the ophthalmic ganglion.
4, On bone close to foramen rotundum, marks the superior maxillary division which is connected below with the spheno-palatine (Meckel's) ganglion, and passes forwards to the infra-orbital foramen.
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6, Chorda tympani. 7, Facial nerve issuing from the stylo-mastoid foramen.

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Where it is due to central causes, trigeminal anæsthesia is never associated with trophic lesions, with vaso-motor, or with secretory disturbances. It is extremely rare also, from centric causes, to meet with any very appreciable paralysis of the masticatory muscles.

Peripheral lesions.—When the root of the fifth nerve is destroyed by disease, situated anywhere between its nucleus in the pons (Fig. 102) and the Gasserian ganglion, there may be complete trigeminal anæsthesia of the same side as well as paralysis of the muscles of mastication; but there would be no trophic, vaso-motor, or secretory troubles. This portion of the nerve is apt to be affected by tumours growing in the pons, when there is also the possibility of an affection of other contiguous nerves at the base of the brain (Fig. 88)—especially of the third, fourth, and sixth. Paralysis of these nerves together with some amount of hemiplegia on the opposite side of the body, constitutes a very typical combination of signs.

Where the Gasserian ganglion itself is either invaded or very severely pressed upon by tumours, we may then get all the remarkable series of defects due to *Complete Paralysis of the Trigeminus*. It will be more convenient to refer to the motor defects first, as all the others are more closely related to one another.

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Where there is mere paresis of these muscles of mastication, the condition is, of course, a little more difficult to recognize; we must judge, however, by touch and by what the patient says as to his ability to chew a crust of bread on the suspected side. It is only with extreme rarity that the motor divisions of the fifth is paralysed simultaneously on both sides. The effect would be that only liquid or semi-fluid food could be taken, and, owing to the relaxation of its muscles, the lower would fall away somewhat from the upper jaw. In these, as well as in the more common cases of unilateral paralysis, the electrical 'reaction of degeneration' may soon show itself in the affected muscles, and may be followed later on by some amount of atrophy.

The tensor palati and the tensor tympani ought to be paralysed as well as the masticatory muscles; but the effects produced are so slight as to be scarcely recognizable. No alteration of the palate on the affected side has been noticed in these cases; and the principal result of paralysis of the tensor tympani seems to be to diminish the patient's power of hearing deep tones on the side implicated.

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The ophthalmic symptoms are particularly noticeable. The acuteness of vision undergoes diminution, partly owing to the lessening of the lachrymal secretion, and partly on account of secondary trophic disturbances and hyperæmia. One of the most interesting trophic disturbances, often occurring with trigeminal anæsthesia, is the so-called 'neuro-paralytic ophthalmia,' in the more serious forms of which we meet with a gradually increasing hyperæmia of the conjunctiva, together with cloudiness and ulceration of the cornea. Ultimately we may have perforation of the cornea and destruction of the eye by panophthalmitis. This is commonly supposed to be due, in accordance with Meissner's view, to the implication of a set of 'trophic fibres' which proceed from the Gasserian ganglion with the ophthalmic division of the fifth nerve. The notion that it is due to the mere loss of sensitivity in the conjunctiva and cornea exposing them unduly to the influence of irritants from without, cannot be regarded as adequate; while the experiments of Sinitzin have shown that paralysis of the vaso-motor nerves supplying these parts, far from being a cause of such trophic lesions, seems, on the contrary, to exercise an influence antagonistic to their progress. Whether these trophic changes of the cornea are due to the damage of special 'trophic nerves' as Meissner supposes, or to peripherally-conducted irritations of its ordinary sensory nerves, seems to be the point principally open to doubt.

Another change in the eyeball that may follow upon trigeminal irritation is simple glaucoma. Donders first called attention to the fact that this condition appeared to be due to irritation of the secretory fibres contained in the trigeminus. It has since been established that irritation either of the trigeminal nucleus in the medulla, or of the peripheral branches of the ophthalmic division—as from the introduction of nicotine into the eye—suffices to produce that increase of intra-ocular pressure (due to increased secretion of the aqueous humour) upon which simple glaucoma depends. Again, increased tension in the globe of the eye, of a temporary and recurrent nature, is met with from time to time in association with some forms of trigeminal neuralgia.

The sense of smell is impaired, but only in consequence of the dryness of the Schneiderian mucous membrane. Owing, however, to the coexisting anæsthesia of the mucous membrane, simple irritation by snuff, ammonia, or other such agents, is no longer capable of producing sneezing when applied merely to the nostril on the affected side.

The sense of hearing can only be affected through paralysis of the tensor tympani, and even then, as already stated, only to a very inconsiderable extent, by causing some diminution in the patient's ability to appreciate deep tones.

The sense of taste was formerly supposed to be either lost or considerably impaired in the anterior two-thirds of the tongue, in cases of severe disease of the trunk of the trigeminus. It is well known that the sense of taste in these parts of the tongue is due to impressions conducted by the lingual nerve; it is, however, equally well known that the so-called gustatory fibres, on their way to the brain, leave the lingual with the chorda tympani, in order to join the facial nerve; and that they certainly continue with it as far as the point of origin of the greater petrosal nerve. The weight of evidence seems now to incline to the view that these gustatory fibres go on to the brain, with the trunk of the facial nerve, as the nerve of Wrisberg. Some have supposed that the gustatory fibres, for the most part at least, leave the facial in the great petrosal nerve, proceeding to Meckel's ganglion (Fig. 104) and thence to the second division of the fifth nerve on their way to the brain. A number of cases have been reported which seem to support this distribution—cases, that is, in which there has been no disease of the facial, but where, with complete trigeminal anæsthesia, there has also been loss of taste in the anterior half of the tongue. Still, it must be borne in mind that nerves, like arteries, are liable to some variation in their distribution; and, therefore, that the gustatory fibres may also at times go to the brain with the facial as the nerve of Wrisberg, as some clinical and experimental data appear to indicate. Cases have been recorded where there has been total paralysis of all three divisions of the fifth, and in which the sense of taste in the anterior half of the tongue has remained intact. Prevost has found, moreover, that the removal of Meckel's ganglion on both sides caused no disturbance of taste; and in a case of the removal of one of Meckel's ganglia, which recently came under my own observation, there was a similar absence of any impairment of taste on the corresponding side. The anatomical investigations of Duval likewise strongly support the view that the gustatory fibres go to the brain with the facial, as the nerve of Wrisberg, and that this nerve, moreover, terminates in a portion of the glosso-pharyngeal nucleus (see p. 447).

After what has been said it would be needless to go into details concerning the effects producible by paralysis of one or other of the three main branches of the sensory division of the fifth. In each case the anæsthesia would be strictly limited to the area of distribution of the branch affected, and with it there might be certain other associated signs to which reference has been made above. Thus, with disease of the third division of the fifth, we should have the association of paralysis of the masticatory muscles; with disease of the second division occurring on the proximal side of the connections with Meckel's ganglion, there might be the association of loss of taste in the anterior half of the tongue on the affected side; while, where the ophthalmic division of the fifth becomes involved in a destructive lesion, there may be the association, with anæsthesia in the parts to which this branch is distributed, of trophic lesions of the eye. The diagnosis in all these cases is simple and likely to be quite free from difficulty.

The mode of diagnosing central and peripheral disease of the fifth nerve has already been referred to (p. 437); and, again, in the case of the existence of peripheral disease, the signs indicative of disease involving the nuclei of the fifth, or the sensory and motor fibres between these nuclei and the Gasserian ganglion, have been pointed out, as contrasted with the signs of destructive disease involving the whole nerve in this latter situation.

We must now refer briefly to the effects of **irritative lesions of the fifth nerve.**

As regards the motor division of the fifth nerve, it may be said that irritative lesions seem only very rarely, if at all, to induce tonic spasms in the muscles of mastication; while clonic spasms in these muscles probably never originate from direct irritation of the motor fibres of the fifth.

Tonic spasm of the masticatory muscles is commonly a bilateral affection and then leads to what is commonly known as trismus or lock-jaw. Where this condition exists, the external masticatory muscles can be felt to be tense and hard. It may exist as part of the condition known as 'tetanus'; or it may occur alone, and in that case has almost always a reflex origin. It may be produced by the action of cold upon some of the superficial branches of the fifth; or it may be due to prolonged irritation operating upon some one of the sensory branches of the fifth, and especially upon the inferior maxillary branch. It is thus an occasional accompaniment ot trigeminal neuralgia. Such irritation may be connected with dentition, with decayed teeth, with injury to the jaw during the extraction of teeth, with tumours of the jaw, or with periostitis. In nervous and susceptible persons, irritation of the intestine or of some other more distant part may, on rare occasions, lead to trismus. In some hysterical patients it may supervene without apparent cause.

Where the spasm is unilateral and partial it is, perhaps, much more likely to be produced by some direct irritation of the motor fibres of the fifth nerve. In these cases of partial spasm the lower jaw may be pulled over to one side, or it may be advanced or retracted.

Clonic spasms of the muscles supplied by the fifth nerve occur as part of the phenomena pertaining to convulsive attacks of different kinds. It occurs also, not unfrequently, in the cold stage of fevers (teeth-chattering), and, generally, during the occurrence of 'rigors.' A 'jaw clonus' has, moreover, been described of late by Beevor, which presented itself in a case of amyotrophic lateral sclerosis.*

A lateral grinding movement of the jaws takes place during sleep in some cases of cerebral disease, and also in some merely nervous persons, especially in epileptics. These clonic spasms of the masticatory muscles therefore, would seem to have no localising value whatever.

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* It would probably often be met with in cases where the 'jaw jerk' is greatly exaggerated. In 'Brain,' Jany., 1886, some details concerning these phenomena are given by Beevor and also by de Watteville. Their significance, if any, for diagnostic purposes has yet to be determined.

As regards the sensory division of the fifth nerve, it is well known that irritative lesions affecting its trunk, and especially the Gasserian ganglion, give rise to a more or less wide spread *neuralgia* over the area of distribution of the nerve (Tic douloureux). The irritation is in other cases, and more frequently, limited to this or that division or branch of the nerve. It is, on the whole, more common in the field of distribution of the first or second, than of the third division of the fifth nerve.

Into the whole natural history of trigeminal neuralgia it would be impossible to enter here. We are principally concerned with it as a concomitant of intra-cranial disease; though there can be little doubt that such instances constitute only a very small proportion of the sum-total of cases of trigeminal neuralgia. Such neuralgias are largely determined by constitutional states or general conditions of the body, and by peripheral irritations either in the territory of the fifth nerve (as from decayed teeth), or in more distant parts—especially when these are of such a nature as to lower the general health. Neuralgias of a very similar type may be induced, at other times, by exposure to wet and cold.

There seem to be good grounds for believing that trigeminal neuralgia is never met with as a result of an affection of the intra-cerebral fibres of the fifth—that is of the fibres which ascend into the hemispheres from the sensory nucleus in the medulla. There appears, on the contrary, to be the best reason for regarding it as a peripheral affection of the fifth nerve—using that word in the widest sense, as inclusive of the medullary nucleus of the nerve. So far as the peripheral branches, or the trunk of the nerve (back as far as and inclusive of the Gasserian ganglion) are concerned, this is very commonly admitted. It is not, however, so commonly recognized that some morbid conditions, causing irritation of the sensory nucleus of the fifth, may also be a cause of neuralgia in the province of this nerve.

Tumours, or aneurysms of the carotid artery, may irritate the trigeminus at the base of the brain, and in these cases it is well to bear in mind the proximity of the motor nerves of the eye-ball (Figs.

type,' still it is most convenient to regard, as *peripheral affections*, only those cases where the nerves are involved outside the cranial cavity. Having excluded the likelihood of intra-cranial lesions, the existence of this or that cause already referred to as possibly operating within the orbit (p. 430) must be considered. In this class of cases, it is well to bear in mind that isolated paralyses of ocular muscles are often complete; while this is rarely so when they are of centric origin.

The squint with possible diplopia, that may be caused by hypermetropia and myopia, must not be confounded with cases in which such symptoms are caused by lesions of nerves supplying the ocular muscles (see p. 148).

D.—THE FIFTH OR TRIGEMINAL NERVE.

This nerve contains a large sensory and a small motor portion. Reference to Figs. 101, 105 (v, v′, v″,) will clearly show the positions

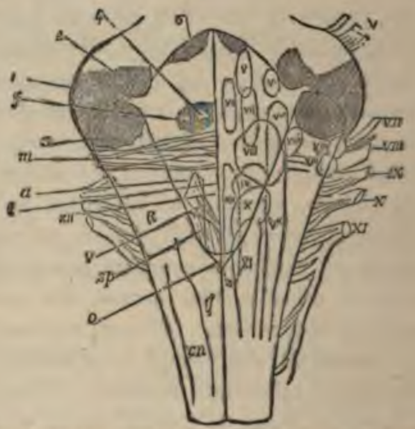

FIG. 101. DIAGRAMMATIC VIEW OF THE POSTERIOR SURFACE OF THE MEDULLA, OR FLOOR OF THE FOURTH VENTRICLE, SHOWING THE RELATIVE POSITIONS OF DIFFERENT NERVE NUCLEI [after Erb].

The left half of the figure represents:—*Cn*, funiculus cuneatus, and *g*, funiculus gracilis; O, obex; *sp*, nucleus of the spinal accessory; *p*, nucleus of the pneumogastric; R, restiform body; XII′, nucleus of the hypoglossal; *t*, funiculus teres; *a*, nucleus of the acousticus; *m*, striæ medullares; 1, 2, 3, middle, superior and inferior cerebellar peduncles. *f*, fovea anterior; 4, eminentia teres (genu nervi facialis); 5, locus cœruleus.

The right half of the figure represents the nerve nuclei diagrammatically:—V, motor trigeminal nucleus; V′, median, and V″, inferior sensory trigeminal nuclei; VI, nucleus of abducens; VII, facial nucleus; VIII, posterior median acoustic nucleus; VIII′, anterior median, VIII″, posterior lateral, VIII‴, anterior lateral acoustic nuclei; IX, glosso-pharyngeal nucleus; X, XI, and XII, nuclei of vagus, spinal accessory, and hypoglossal nerves respectively. The Roman numerals at the side of the figure from V to XI represent the corresponding nerve roots.

in the pons and medulla of the sensory and motor nuclei of this nerve, and their relations with other nerve nuclei. The sensory and motor fibres in connection with these nuclei, appear, as is well known, at the surface near the middle of each lateral region of the pons. In the natural position of the body the small motor portion lies underneath the sensory division, passing beneath the Gasserian ganglion (Fig. 88), it then immediately joins the third or infra-maxillary branch of this division of the fifth. The other two branches—viz., the ophthalmic and the superior maxillary—are, therefore, purely sensory nerves.

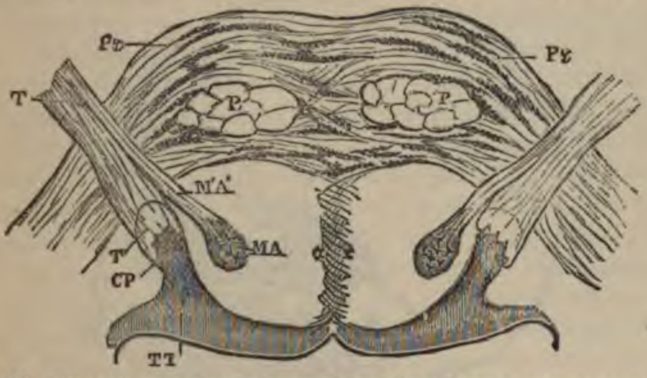

FIG. 102. DIAGRAM OF A SECTION THROUGH THE PONS AT THE LEVEL OF THE EMERGENCE OF THE FIFTH NERVES [after M. Duval].

P,P, Pyramidal strands. Pr, Transverse fibres of the pons with interstratified masses of grey matter. TT, Grey substance of the floor of the fourth ventricle (locus cœruleus). CP, Gelatinous substance of Rolando. T, Ascending root of the trigeminus, curving so as to emerge as part of the larger root of the trigeminus. MA, Motor nucleus of the trigeminus. M'A', Motor root of the trigeminus, (masticatory nerve). T', The Fifth Nerve at its point of emergence from the pons.

It is important to bear in mind that the third, the fourth, and the sixth nerves are, at the base of the brain, close to one another and to the fifth nerve ; and, moreover, that they pass through the dura mater at a very small distance from one another (Figs. 88 and 99).

Lesions of the fifth nerve are apt to produce varied effects, partly sensory, partly motor, while they may also entail certain disorders of secretion and of nutrition. Secretion is affected, because the nerve contains fibres which proceed to the lachrymal, parotid and submaxillary glands. Nutrition is impaired for a reason which is not as yet beyond the reach of doubt. It is very commonly supposed that the nerve contains certain special trophic fibres which have their origin in the Gasserian ganglion ; and that the injury of these fibres accounts for the occurrence of inflammations of the eyeball, ulcerations of the cornea, and also ulcerations of the mucous membrane of the mouth—effects that are undoubtedly apt to occur in association with some diseases of the fifth nerves. It seems possible, however, that these effects may be produced by means of irritations transmitted to the tissues in a centrifugal direction, through the ordinary sensory fibres of the nerves—that they may, in fact, be effects occurring quite independently of the existence of any special trophic fibres. *Vaso-motor fibres* for the conjunctiva and the iris are also distributed with the trigeminus.

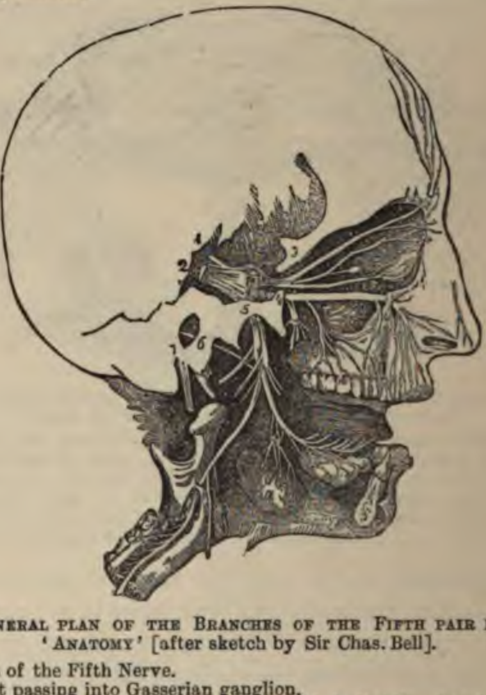

FIG. 103. GENERAL PLAN OF THE BRANCHES OF THE FIFTH PAIR FROM QUAIN'S 'ANATOMY' [after sketch by Sir Chas. Bell].

1, small root of the Fifth Nerve.
2, Large root passing into Gasserian ganglion.
3, Placed on bone above ophthalmic nerve, which is dividing into frontal, lachrymal, and nasal branches, the latter connected with the ophthalmic ganglion.
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The sense of smell is impaired, but only in consequence of the dryness of the Schneiderian mucous membrane. Owing, however, to the coexisting anæsthesia of the mucous membrane, simple irritation by snuff, ammonia, or other such agents, is no longer capable of producing sneezing when applied merely to the nostril on the affected side.

The sense of hearing can only be affected through paralysis of the tensor tympani, and even then, as already stated, only to a very inconsiderable extent, by causing some diminution in the patient's ability to appreciate deep tones.

The sense of taste was formerly supposed to be either lost or considerably impaired in the anterior two-thirds of the tongue, in cases of severe disease of the trunk of the trigeminus. It is well known that the sense of taste in these parts of the tongue is due to impressions conducted by the lingual nerve; it is, however, equally well known that the so-called gustatory fibres, on their way to the brain, leave the lingual with the chorda tympani, in order to join the facial nerve; and that they certainly continue with it as far as the point of origin of the greater petrosal nerve. The weight of evidence seems now to incline to the view that these gustatory fibres go on to the brain, with the trunk of the facial nerve, as the nerve of Wrisberg. Some have supposed that the gustatory fibres, for the most part at least, leave the facial in the great petrosal nerve, proceeding to Meckel's ganglion (Fig. 104) and thence to the second division of the fifth nerve on their way to the brain. A number of cases have been reported which seem to support this distribution—cases, that is, in which there has been no disease of the facial, but where, with complete trigeminal anæsthesia, there has also been loss of taste in the anterior half of the tongue. Still, it must be borne in mind that nerves, like arteries, are liable to some variation in their distribution; and, therefore, that the gustatory fibres may also at times go to the brain with the facial as the nerve of Wrisberg, as some clinical and experimental data appear to indicate. Cases have been recorded where there has been total paralysis of all three divisions of the fifth, and in which the sense of taste in the anterior half of the tongue has remained intact. Prevost has found, moreover, that the removal of Meckel's ganglion on both sides caused no disturbance of taste; and in a case of the removal of one of Meckel's ganglia, which recently came under my own observation, there was a similar absence of any impairment of taste on the corresponding side. The anatomical investigations of Duval likewise strongly support the view that the gustatory fibres go to the brain with the facial, as the nerve of Wrisberg, and that this nerve, moreover, terminates in a portion of the glosso-pharyngeal nucleus (see p. 447).

After what has been said it would be needless to go into details concerning the effects producible by paralysis of one or other of the three main branches of the sensory division of the fifth. In each case the anæsthesia would be strictly limited to the area of distribution of the branch affected, and with it there might be certain other associated signs to which reference has been made above. Thus, with disease of the third division of the fifth, we should have the association of paralysis of the masticatory muscles; with disease of the second division occurring on the proximal side of the connections with Meckel's ganglion, there might be the association of loss of taste in the anterior half of the tongue on the affected side; while, where the ophthalmic division of the fifth becomes involved in a destructive lesion, there may be the association, with anæsthesia in the parts to which this branch is distributed, of trophic lesions of the eye. The diagnosis in all these cases is simple and likely to be quite free from difficulty.

The mode of diagnosing central and peripheral disease of the fifth nerve has already been referred to (p. 437); and, again, in the case of the existence of peripheral disease, the signs indicative of disease involving the nuclei of the fifth, or the sensory and motor fibres between these nuclei and the Gasserian ganglion, have been pointed out, as contrasted with the signs of destructive disease involving the whole nerve in this latter situation.

We must now refer briefly to the effects of **irritative lesions of the fifth nerve.**

As regards the motor division of the fifth nerve, it may be said that irritative lesions seem only very rarely, if at all, to induce tonic spasms in the muscles of mastication; while clonic spasms in these muscles probably never originate from direct irritation of the motor fibres of the fifth.

Tonic spasm of the masticatory muscles is commonly a bilateral affection and then leads to what is commonly known as trismus or lock-jaw. Where this condition exists, the external masticatory muscles can be felt to be tense and hard. It may exist as part of the condition known as 'tetanus'; or it may occur alone, and in that case has almost always a reflex origin. It may be produced by the action of cold upon some of the superficial branches of the fifth; or it may be due to prolonged irritation operating upon some one of the sensory branches of the fifth, and especially upon the inferior maxillary branch. It is thus an occasional accompaniment ot trigeminal neuralgia. Such irritation may be connected with dentition, with decayed teeth, with injury to the jaw during the extraction of teeth, with tumours of the jaw, or with periostitis. In nervous and susceptible persons, irritation of the intestine or of some other more distant part may, on rare occasions, lead to trismus. In some hysterical patients it may supervene without apparent cause.

Where the spasm is unilateral and partial it is, perhaps, much more likely to be produced by some direct irritation of the motor fibres of the fifth nerve. In these cases of partial spasm the lower jaw may be pulled over to one side, or it may be advanced or retracted.

Clonic spasms of the muscles supplied by the fifth nerve occur as part of the phenomena pertaining to convulsive attacks of different kinds. It occurs also, not unfrequently, in the cold stage of fevers (teeth-chattering), and, generally, during the occurrence of 'rigors.' A 'jaw clonus' has, moreover, been described of late by Beevor, which presented itself in a case of amyotrophic lateral sclerosis.*

A lateral grinding movement of the jaws takes place during sleep in some cases of cerebral disease, and also in some merely nervous persons, especially in epileptics. These clonic spasms of the masticatory muscles therefore, would seem to have no localising value whatever.

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* It would probably often be met with in cases where the 'jaw jerk' is greatly exaggerated. In 'Brain,' Jany., 1886, some details concerning these phenomena are given by Beevor and also by de Watteville. Their significance, if any, for diagnostic purposes has yet to be determined.

As regards the sensory division of the fifth nerve, it is well known that irritative lesions affecting its trunk, and especially the Gasserian ganglion, give rise to a more or less wide spread *neuralgia* over the area of distribution of the nerve (Tic douloureux). The irritation is in other cases, and more frequently, limited to this or that division or branch of the nerve. It is, on the whole, more common in the field of distribution of the first or second, than of the third division of the fifth nerve.

Into the whole natural history of trigeminal neuralgia it would be impossible to enter here. We are principally concerned with it as a concomitant of intra-cranial disease; though there can be little doubt that such instances constitute only a very small proportion of the sum-total of cases of trigeminal neuralgia. Such neuralgias are largely determined by constitutional states or general conditions of the body, and by peripheral irritations either in the territory of the fifth nerve (as from decayed teeth), or in more distant parts—especially when these are of such a nature as to lower the general health. Neuralgias of a very similar type may be induced, at other times, by exposure to wet and cold.

There seem to be good grounds for believing that trigeminal neuralgia is never met with as a result of an affection of the intra-cerebral fibres of the fifth—that is of the fibres which ascend into the hemispheres from the sensory nucleus in the medulla. There appears, on the contrary, to be the best reason for regarding it as a peripheral affection of the fifth nerve—using that word in the widest sense, as inclusive of the medullary nucleus of the nerve. So far as the peripheral branches, or the trunk of the nerve (back as far as and inclusive of the Gasserian ganglion) are concerned, this is very commonly admitted. It is not, however, so commonly recognized that some morbid conditions, causing irritation of the sensory nucleus of the fifth, may also be a cause of neuralgia in the province of this nerve.

Tumours, or aneurysms of the carotid artery, may irritate the trigeminus at the base of the brain, and in these cases it is well to bear in mind the proximity of the motor nerves of the eye-ball (Figs.

88 and 99), since the coincidence of neuralgia in certain parts of the territory of the fifth nerve with paresis or paralysis of two, or perhaps all three, of the motor nerves of the eye on the same side, are signs of great localising value.

Again, thickenings, syphilitic or other, of the trunk of the nerve or of one of its main branches may occur within the skull. Or during their passage through the foramina of exit, one or other of the divisions of the nerve may be compressed. Outside the skull either of the branches may, again, be involved in tumours; or those of the second and third divisions may be compressed or invaded by surrounding pathological conditions as they pass through canals in the upper or lower maxillary bones.

The more distinctly the pain is limited to the territory of one particular branch of the fifth nerve, the greater is the probability that it is due to peripheral irritation affecting some part of that branch.

Some amount of hyperalgesia is often present in the neuralgic territory, leading to an abiding tenderness when the part is touched. Such a condition must not be confounded with inflammation of the periosteum of the facial bones, or of the lining membrane of the antrum or of the frontal sinuses. The photophobia which is often associated with neuralgia of the first division of the fifth is commonly supposed to be due to a hyperalgesic condition of the branches distributed to the conjunctiva. In this condition there is also, as a rule, increased lachrymation instead of the diminished lachrymation that goes with paralysing lesions of the ophthalmic division of the fifth.

Similarly, with neuralgia of the second division of the fifth, there may be a great increase in the amount of the secretion from the nasal and buccal mucous membranes, and at times also from the salivary gland, as well as increased lachrymation, brought about in a reflex manner.

Again, it must be borne in mind that neuralgiæ of different branches of the fifth nerve are specially apt to be associated with spasms of some of the muscles innervated by the facial nerve; and that vaso-motor and trophic disturbances of various kinds are apt to be met with in the neuralgic areæ:—such as undue pallor or redness of the skin, changes in the colour or texture of the hair, neuro-paralytic ophthalmia, a proneness to inflammation on slight provocation in other areæ, the occurrence of herpetic or other skin eruptions, an overgrowth of epithelium on the corresponding half of the tongue where the third division of the nerve is affected, or the occurrence of iritis or glaucoma when there is neuralgia of the ophthalmic division of the fifth.

E.—THE FACIAL NERVE.

Paralysis of the facial nerve may be due to central (intra-cerebral), or to peripheral (extra-cerebral) causes.

The facial paralyses that belong to the first category have already been fully considered (pp. 171, 339). We need only say here, therefore, that facial paralyses of central origin are usually associated with a more or less distinct hemiplegic condition; that they are characterised by normal electrical reactions, by the persistence of reflex and associated movements, and by the exemption from paralysis of certain muscles in the upper half of the face, viz., the occipito-frontalis, the corrugator supercilii, the orbicularis palpebrarum, and the dilator naris.

Where the paralyses of the facial nerve belong to the second primary division, and are of peripheral (or extra-cerebral) origin, we may, on the other hand, look for the following broad features:—an absence of the hemiplegic condition; the existence in all severe cases of decidedly abnormal electrical reactions; the loss of reflex and associated movements; and, lastly, the existence of paralysis in the upper as well as in the lower half of the face.

It is this latter type of paralysis with which we are now concerned. It is commonly spoken of as **Bell's Paralysis,** or **Facial Palsy.** It is extremely frequent and may proceed from the most diverse causes, owing to the long and varied course pursued by the facial nerve in its passage from its nuclear origin in the pons Varolii, outwards to its terminal distribution in the muscles of the face. In any given case of this extra-cerebral form of facial palsy we have, in fact, to decide whether the nerve is affected

(*a*).—Within the Cranium.
(*b*).—In the Fallopian Canal.
(*c*).—Outside the Cranium.

Before giving a synopsis of the signs indicative of paralysis of the facial nerve in this or that region above mentioned, it is essential that attention should be briefly called to a few anatomical and physiological facts concerning the branches of the facial and their relations with other nerves, with glands, and with muscles.

Innervation of the palate.—From the geniculate swelling on the facial nerve where it changes its direction, near the bottom of the internal auditory canal, the great superficial petrosal nerve is given off, which passes through a canal in the temporal bone to Meckel's ganglion, and thence, it is said, to the levator palati muscle. When this nerve is paralysed, it is commonly thought that the arch of the palate is lowered on the corresponding side, that during phonation it is distinctly drawn towards the opposite side; and that there is also an absence of reflex contraction when the palate is touched on the paralysed side. On this subject, however, there is room for very grave doubt, if not actual denial. It is certainly extremely rare to meet with any such results as consequences of disease in the intra-cranial portion of the facial nerve. Moreover, in a recent case in which Meckel's ganglion was completely removed by V. Horsley from one of Hughlings Jackson's patients, I satisfied myself, by an examination, made about three weeks after the operation, that the palate was quite symmetrical and its mobility on the side on which Meckel's ganglion had been removed not in the least impaired. Concerning the nerves which do supply the palate, see under H.—The Pneumo-gastric Nerves.

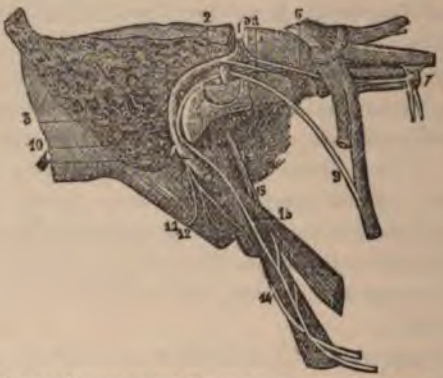

FIG. 104. THE FACIAL NERVE IN ITS CANAL WITH ITS CONNECTING BRANCHES [from Sappey after Hirschfeld and Leveillé].

The mastoid and a part of the petrous bone have been divided nearly vertically, and the canal of the facial nerve (Fallopian) opened in its whole extent from the internal meatus to the stylo-mastoid foramen; the Vidian canal has also been opened from the outside.

1, Facial nerve in the first horizontal part of its course; 2, its second part turning backwards; 3, its vertical portion; 4, the nerve at its exit from the stylo-mastoid foramen.

5, Geniculate ganglion. 6, Large superficial petrosal nerve.

7, Spheno-palatine (Meckel's) ganglion. 8, Small superficial petrosal nerve. 9, Chorda tympani. 10, Posterior auricular branch cut short. 11, Branch to the digastric muscle. 12, Branch to the stylo-hyoid muscle. 13, Twig uniting with the glosso-pharyngeal nerve (14 and 15).

The uvula sometimes deviates to one side and sometimes to another; its innervation is probably variable. Even in conditions of health deviation of the uvula to one or other side may be met with. Its position does not, therefore, afford any reliable indication in cases of paralysis.

The *Chorda tympani* contains nerves of taste for the anterior half or two-thirds of the tongue, and also secretory nerves that go to several of the salivary glands. The secretory nerves leave the brain with the facial and then distribute themselves as part of the chorda tympani. The gustatory fibres, however, whilst they reach the facial nerve with the chorda tympani, were generally supposed to remain as part of this trunk only as far as the geniculate ganglion and then to leave it with the greater petrosal nerve; consequently it was supposed to be only in disease of this part of the facial nerve that the sense of taste in the anterior part of the tongue became lost or impaired, on the affected side, to acid, saline, or sweet substances. The recent anatomical researches of Duval tend, however, to show that the gustatory fibres of the chorda tympani do not pass off with the greater petrosal to join the fifth nerve, but that they continue with the facial as the nerve of Wrisberg which, as he shows, terminates in a portion of the nucleus of the glosso-pharyngeal nerve. I have met with one well-marked case of disease in the intra-cranial portion of the facial nerve in which the sense of taste in the anterior part of the tongue on the same side was lost, thus tending to confirm Duval's view. The evidence against the previously entertained view, that the gustatory fibres of the chorda tympani leave the facial, and go by way of Meckel's ganglion to the fifth nerve, has already been given (p. 440).

It is only some of the *secretory nerves* of the facial that are distributed with the chorda tympani; others leave the facial higher up with the lesser petrosal nerve, on their way to the otic ganglion. Disease of the facial on the proximal side of the lesser petrosal nerve causes, therefore, the most marked dryness of the mouth from diminution of the salivary secretion; though this sign may be met with, to a lesser degree, when the facial nerve is affected lower down—that is, till we reach the point where the chorda tympani leaves the facial, about one-fourth of an inch above the stylo-mastoid foramen.

It should be remembered that the *sense of smell* is sometimes distinctly impaired, on the affected side, in cases of facial paralysis, this being due to two causes: first, from dryness of the corresponding side of the nose, owing to the fact that tears flow to an unnatural extent over the side of the face; and, secondly, because the paralysis of two of the muscles of the nostril (the levator alæ nasi and the compressor naris) interferes with the proper drawing in of air to the olfactory region of the nose.

When the branch of the facial nerve which supplies the *stapedius muscle* is affected, there is, as a consequence, an over-action of the tensor tympani muscle, leading to abnormal sensitiveness to all musical tones or an abnormal power of perceiving deep notes. If, therefore, in cases of paralysis of the facial nerve, we meet with evidence of an unwonted acuteness of hearing of any kind, it would be an indication that the nerve was affected at some point above the point at which the branch to the stapedius is given off.

The detailed objective signs to be met with in cases of peripheral paralysis of the facial nerve will be given presently. It will be well first to look to the indications afforded by the anatomical facts above stated and to others, for the determination of the particular part of the nerve that is affected in any given case of facial palsy.

Signs of Affection of the Facial Nerve within the Cranium.—Under this head we include the course of the facial from its nucleus in the pons (near the middle line, beneath the floor of the fourth ventricle) to the surface of the medulla, and thence as far as the bottom of the internal auditory meatus. When the nucleus, or the root of the nerve near it, is affected, the sixth nerve, whose fibres wind round the nucleus of the facial (Fig. 105), is apt to be simultaneously paralysed, and there would also frequently be some amount of hemiplegia on the opposite side of the body. At the surface of the base of the brain, any disease that implicates the facial is likely also to involve the auditory nerve, consequently there may be the association of sub-

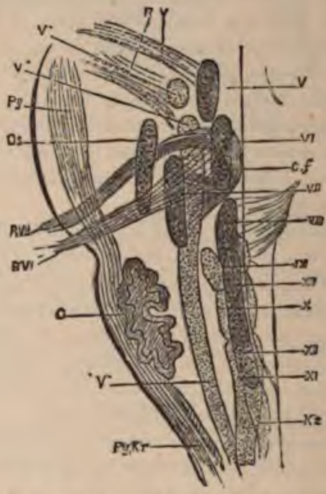

FIG. 105. TRANSPARENT LATERAL VIEW OF THE MEDULLA, SHOWING DIAGRAMMATICALLY THE RELATIVE POSITIONS OF THE MOST IMPORTANT NERVE NUCLEI [after Erb].

The right half of the Medulla seen from the surface of section; the parts that lie closer to this surface are most deeply shaded.

Py, pyramidal tract; Py, Kr, decussation of pyramids. O, olivary body; Os, superior olivary body; V, motor; V', middle sensory; V, inferior sensory nucleus of trigeminus; VI, nucleus of abducens; Gf, genu facialis nervi; VII, nucleus facialis; VIII, posterior median acoustic nucleus; IX, glosso-pharyngeal nucleus; X, nucleus of vagus; XI, nucleus of the accessorius; XII, hypoglossal nucleus; Kz, nucleus of the funiculus gracilis; Rv, trigeminus root; RVI, root of the abducens; R VII, root of the facialis.

jective sounds of different kinds, or more or less of actual deafness. Extreme dryness of the side of the mouth, is also a sign of implication of the nerve in this part of its course. The effects that would otherwise follow from paralysis of the stapedius muscle, may be non-apparent, owing to some coincident diminution of function in the auditory nerve itself. Paralysis of the palate on the side on which the face is paralysed is, as we have already pointed out, not to be looked for, though formerly it was commonly regarded as indicative of a lesion involving the nerve above the geniculate ganglion. Loss of taste in the anterior part of the tongue may, on the contrary, be expected to occur.

Signs of Affection of the Facial Nerve within the Fallopian Canal.—Where the disease involves the geniculate ganglion, in addition to complete paralysis of the face, there is pretty certain to be extreme dryness of the mouth on the same side, loss of taste in the anterior half of the tongue, and also increased sensitiveness to musical sounds.

With damage to the nerve slightly below the geniculate ganglion, the facial palsy would be associated only with moderate dryness of the mouth on the affected side, loss of taste in the anterior half of the tongue, together with increased sensitiveness to musical sounds.

Below the giving off of the nerve to the stapedius muscle, and thence downwards to one-fourth of an inch from the stylo-mastoid foramen (where the chorda tympani leaves the facial), compressing lesions of the facial nerve cause, in addition to facial paralysis, only moderate dryness of the mouth on the affected side together with loss of taste in the anterior half of the tongue.

Signs of Affection of the Facial Nerve outside the Cranium.—When disease affects the trunk of the nerve in the lower extremity of the Fallopian canal, after the giving off of the chorda tympani, the effects are, for all practical purposes, to be regarded as similar to those resulting from compression or disease of the facial outside the skull. We have in these cases to do with paralyses of muscles only; there is no longer dryness of the mouth or impairment of taste on the paralysed side. As in all cases of 'Bell's paralysis,' however, there may be some amount of impairment of the sense of smell, owing to causes already referred to.

Where the trunk of the facial is involved outside the Fallopian canal the paralysis is apt to be less absolute than when the nerve becomes the seat of disease, inflammatory or other, within the unyielding walls of this canal. In such cases, also, where there is less paralysis and less damage to the fibres of the nerve, the electrical reactions may be only slightly abnormal.

The paralysis that occurs in the area of distribution of the facial nerve in a well-marked case of facial palsy or 'Bell's paralysis' presents the following combination of characters:—

The paralysed side of the face is smooth and expressionless; when at rest, it may be seen that the side of the forehead especially is smoother, that the eye may be slightly more open, and that the angle of the mouth is lower and the lip less arched on the paralysed side. The patient cannot wrinkle his forehead, raise his eyebrow, or close his eye; when he attempts to perform this latter act, the eyelids are not approximated but the eyeball rolls upwards and inwards and the eye consequently remains open during sleep.) Winking is produced neither by directing a finger towards the eye, nor by actually touching the conjunctiva. The permanently open state of the eye exposes it unduly to irritation from foreign particles; hence it is apt to appear bloodshot or slightly inflamed. Owing to the paralysis of Horner's muscle the tears run over the cheek instead of entering the lachrymal canal. The nostrils on the two sides are often unequal; and that on the paralysed side, instead of expanding during inspiration, falls in. The tip of the nose is sometimes drawn over towards the healthy side. The angle of the mouth cannot be raised on the paralysed side, and when the patient talks, laughs, or cries, that of the healthy side is notably drawn up, owing to the unbalanced action of the corresponding zygomatici. The buccinator is flaccid, and is apt to be puffed out during speaking or coughing; food collects between the jaws and the cheek; and the patient is unable to whistle or blow out a candle, owing to imperfect apposition of the lips on the paralysed side. Saliva is also apt to dribble away on the same side.

It is almost needless to refer to other paralyses in the sphere of the facial nerve, because they are comparatively unimportant, and often quite escape detection; still, it should be borne in mind that the muscles of the external ear, as well as the platysma, the posterior belly of the digastric, and the stylo-hyoid muscle, are all innervated by the facial nerve.

The electrical reactions in 'Bell's Paralysis' have been most carefully studied by Erb. He considers that all cases may be ranged into one or other of three categories:—First, slight cases in which the electrical reactions do not differ from the normal; secondly, severe cases in which a typical 'reaction of degeneration' (see p. 10) may present itself in from ten to fourteen days from the date of onset; thirdly, a great number of intermediate cases, in which the 'reaction of degeneration' becomes very incompletely developed. In this latter group of cases, it happens, not unfrequently, that the muscles show the characteristic features of the reaction of degeneration, although the nerve shows it only to an incomplete degree—that is to say, its excitability to both currents is diminished but not lost (showing that the nerve has not undergone complete degeneration).

PATHOLOGICAL DIAGNOSIS.

The pathological diagnosis is often a matter of considerable difficulty, owing to the very varied nature of the causes that may produce facial paralysis. At other times the diagnosis is easy, because of the frequency of two sets of causes in particular, and of the existence of strong evidence that one or other of them has been in operation. The first set of such causes includes the cases in which there has been prolonged exposure to cold, especially to cold draughts of air playing on one side of the face, as from sitting in a railway carriage on a cold day by the side of an open window, or by falling asleep near an open window. In the second set of these frequently recurring causes, are found cases where some chronic suppurative inflammation exists in the middle ear, and in which an inflammatory condition spreads to, or the pressure of inflammatory products operates upon, the contiguous facial nerve.

In all cases, it is important first to arrive at a regional diagnosis, as to the precise part of its course in which the facial nerve is implicated, since, in this way, the pathological diagnosis is often much simplified. With a view to the accomplishment of this end, we now subjoin summaries of the principal pathological conditions by which the facial nerve is most likely to become implicated in the three parts of its course respectively.

Within the Cranium.—Hæmorrhage, softening, or new growth may involve the nuclei or root fibres of the facial nerve, beneath the floor of the fourth ventricle or in the pons (Figs. 75, 79). The nerve itself may become involved by contiguous chronic meningitis; its sheath may become thickened or the seat of new growths (either of syphilitic or of non-syphilitic origin); or the nerve may be implicated by some new growth springing either from the dura mater or from the temporal bone.

Within the Fallopian Canal.—Fractures of the temporal bone, caries, suppurative disease of the middle ear, syphilitic disease of the temporal bone or of the nerve itself, or involvement of the nerve by tumours in the temporal bone, may all act as causes of facial paralysis from an affection of the nerve in this part of its course. It seems very doubtful whether the facial nerve can be primarily affected within the Fallopian Canal by the neuritis that follows exposure to cold, although it undoubtedly extends to this part of the nerve secondarily.

Outside the Cranium.—Exposure to cold, producing the so-called 'rheumatic inflammation' of the sheath of the nerve; traumatic causes, such as gunshot or punctured wounds of various kinds; pressure upon or involvement of the nerve by tumours of the parotid gland or abscesses in its neighbourhood, are the commonest causes of disease of the facial in this part of its course.

Lastly, it is worth mentioning that in cases of instrumental labour, facial paralysis has at times been produced by some undue pressure upon the nerve caused by the forceps. It is important also to note, in this connection, that in infants and very young children there is often no appreciable lack of symmetry about the face so long as it is at rest; but when the child laughs or cries the facial paralysis at once becomes obvious, more especially by the apparently extreme drawing up of the opposite side of the mouth.

Double facial paralysis (Diplegia Facialis) is a rare condition that requires only a very brief notice. The face is here absolutely fixed and expressionless, but, owing to the bilateral nature of the affection, there is no lack of symmetry about the mouth and the nose.

The condition may be due to different kinds of pathological causes acting upon the nerves, either within the cranium or else within the Fallopian canals. If caused by disease occurring simultaneously in both temporal bones, both facial nerves may be paralysed and taste may be lost in the anterior two-thirds of the tongue. Should it be due, however, to intra-cranial disease in the form of a chronic meningitis, other contiguous cranial nerves generally become implicated, and such associations tend to throw light upon the seat of the disease. This may also occur where syphilitic thickenings or growths at the base of the brain act as causes of a double facial paralysis. Again, there may be a similar association of paralysis of other cranial nerves (such as the hypoglossal, the spinal accessory, or the auditory), where diplegia facialis is caused by progressive degenerative disease of the medulla, or by small focal lesions or tumours beneath the door of the fourth ventricle, where many nerve nuclei lie so close together (Fig. 105).

Spasms of the Muscles supplied by the Facial Nerve.

Some few remarks must be made on this subject, on account of the relationship of spasms to paralyses of muscles supplied by the facial nerve.

Spasms of the facial muscles commonly occur on one side, though they may at times be bilateral. Again they may affect all the muscles of one side of the face, the diffuse form; or they may affect one or two muscles only, the partial form of the disease.

Diffuse spasm of the facial muscles, or the true convulsive tic, occurs in the form of clonic convulsions, and is the variety most frequently met with. The spasms occur in paroxysms lasting from two seconds to two minutes; and these paroxysms recur with very varying frequency, perhaps only one or two attacks in the twenty-four hours, though in other cases they may, for a time at least, recur with extreme frequency—even twenty to forty attacks in an hour have been recorded. The attacks are neither associated with any loss of consciousness nor with pain; they consist of mobile and changing spasms, now of this, now of that facial muscle—often beginning gradually and subsiding in the same manner. In rare cases the spasm extends, so as to involve the opposite side of the face. Sometimes, moreover, it extends so as to involve the muscles supplied by other nerves—especially to the muscles of mastication and to those of the tongue. Occasionally, even the muscles of the neck and shoulders have been known to share in the spasm.

During the intervals between the attacks, voluntary control over the muscles is found to be in no way impaired; while the electrical reactions, both of the facial nerve and of its muscles, may also be quite natural.

Tonic spasm of the facial muscles, in its diffuse form is a condition of very great rarity. Erb, indeed, expresses a strong opinion that most of these cases "are only those forms of muscular contracture which so commonly occur in the later stages of severe facial paralysis, and with which clonic contractions of the facial muscles are subsequently so often associated." He believes that only a very few cases are due to other unknown causes.

In regard to the causation of convulsive tic nothing sufficiently definite has yet been ascertained. We do not know positively whether it is most apt to be associated with some irritating lesions affecting the intra-cerebral fibres of the facial nerves or their cortical end stations; or whether it is more frequently produced by some irritation affecting the nucleus of the facial nerve in the lower part of the pons. It would seem quite unlikely that it should be caused by irritation acting directly upon any part of the facial nerve itself; although in a large proportion of the cases its onset is reported to have had some connection with exposure to cold. In reference to this, the above-quoted view of Erb must not be forgotten. Evidence exists, however, tending to show that it may be caused by disease contiguous to the lower and anterior part of the Rolandic area; and, still more frequently, by pathological processes irritating the nuclei of the facial nerve.

Violent emotions, either of anger or fear, sometimes act as exciting causes of convulsive tic; while still more often it seems to be determined by some reflex irritations. In both sets of cases we should probably have to do with an unnatural nutritive, as well as functional, condition of the nucleus of the facial, on the side affected. In the latter class, the seat of irritation is usually to be found in some part of the area of distribution of the fifth nerve. In other cases, however, the irritations have been supposed to start from worms or some such sources of irritation within the alimentary canal, or to be consequences of one or other form of uterine disease.

Of the *partial forms of facial spasm* those that affect the eyelids are by far the most frequent, and also the most important.

Tonic spasm of the eyelids, commonly known as blepharospasm, is produced by contraction of the orbicularis palpebrarum, which may be either persistent for weeks or months, or may recur in paroxysms lasting either for minutes or for hours. It may exist in one eye alone or in both. In a large proportion of the cases it seems to have a reflex origin, and is especially frequent as a consequence of ulceration of the cornea or inflammation of the conjunctiva. At other times, the source of irritation may be in some other part of the area of distribution of the fifth nerve.

Clonic spasm in the eyelids (nictitatio) is met with only with extreme rarity. It may occur alone or in association with blepharospasm, and seems to be commonly induced in much the same way.

Other partial spasms of little diagnostic or practical importance occur, at times, in the zygomatici, or in the corrugator supercilii.

F.—THE AUDITORY NERVE.

The fibres of the auditory nerves arise, on each side, from four nerve nuclei situated in the floor of the fourth ventricle, whose position with regard to one another and the nuclei of other nerves is shown in Fig. 101, viii-viii‴. Its fibres appear at the surface on the outer side of the medulla, in the groove between that body and the pons, just outside those of the facial nerve (Fig. 87). From this point, these two nerves pass almost horizontally outwards, and after a very short course enter the internal auditory meatus.

The short course of the auditory nerves from their origin in the medulla to the internal ear, and their protected condition within the internal auditory canal, contrasts markedly with the much longer and less protected course taken by the optic tracts and nerves between their nuclei of origin and the eyeballs. There is an almost similar disproportion between the frequency of the implication of the sense of hearing and the sense of sight respectively, as a consequence of intra-cranial disease: the former being comparatively rare and the latter common. In each case the impairment of these two special senses by intra-cranial disease is due principally to morbid processes implicating either their nerve fibres in some part of their course, or their respective nuclei. Grave impairment of these senses is much less frequently caused by intracerebral lesions that do not involve either the root fibres or their nuclei of origin. All that is needful to be said concerning unilateral deafness caused by such lesions may be found under the head of Hemianæsthesia (p. 153), since such defects, whether from structural or from functional disease of the brain usually do not occur alone but in common with lack of other modes of sensibility, both general and special, on the same side of the body.

Again, it may be well to call attention to the fact that in cases of intra-cranial tumour, there is, so far as we know, no frequent affection of the organs of hearing at all comparable with optic neuritis, commonly double and often leading, as it does, to notable diminution or even loss of sight. This is, indeed, one of the reasons why hearing is less frequently affected in intra-cranial disease than the sense of sight; and we may find in it also an explanation of the fact that the impairment of hearing due to intra-cranial disease is less apt to be bilateral than impairment of sight from the same causes.

In trying to estimate the significance of partial or complete deafness, unilateral or bilateral, the first question to be considered is, whether such defect is of primary nervous origin, or whether it is due to disease of the middle ear, which is such a very common cause of deafness. In settling this primary diagnostic problem, regard must be had to the patient's previous history and to the existence or not, past or present, of discharge of a purulent character from the external ear; to the duration of deafness; to the nature of any concomitant nervous symptoms, and to the question, if any such symptoms exist, as to their order of development—that is, whether after, simultaneously with, or before the development of the deafness —so far as such information can be obtained. Information of the kind last referred to is often to be had only in a vague and uncertain manner. Patients are frequently found to be more or less deaf on one side, who will tell you themselves that their hearing is quite good; and such unilateral partial deafness on one side is undoubtedly very common in this country, as a sequence of mere slight catarrhal inflammations of the middle ear—that is, in patients in whom no suppurative disease of the middle ear associated with discharge from the external meatus has ever occurred. Again, in other cases, whether the patient is or is not aware of a partial unilateral or bilateral deafness, such defect may, on examination, be found to exist and be ascertained to be dependent upon an accumulation of cerumen, or wax, in the external meatus. Thus, in the investigation of all cases of deafness, the external meatus must be carefully examined by the aid of the ear speculum, to ascertain whether such deposits or other obstructions in the form of tumours or polypi exist within the external auditory canal; or, in the absence of any of such causes, to ascertain whether the membrana tympani is, or is not, intact.

Beyond the enquiries and methods suggested above, it is right to have recourse to another means to help us to decide whether the deafness is primarily of nervous origin, or whether it is due to some chronic inflammatory disease of the middle ear, past or present. This additional mode of examination is the testing of the patient's ability to appreciate the vibrations of the tuning fork through the bones of the skull, when, after having been struck, its extremity is placed upon some part of the scalp, either in the middle line or towards one or other ear. In cases of primary nervous deafness the ability to hear the tuning fork through the bones of the skull on the affected side is decidedly diminished. On the other hand, in cases of disease of the middle ear, although the vibrations of the tuning fork are heard much less distinctly on the affected side, when the instrument is held alternately at a slight distance away from either meatus, they are, on the contrary, when the instrument is applied to the scalp in the middle line over the forehead or vertex, actually heard with more distinctness on the affected than on the non-affected side. Although rather difficult to explain satisfactorily, there can be no doubt that this latter peculiarity exists in the great majority of cases, and, therefore, it often proves of valuable diagnostic importance in cases of unilateral deafness of doubtful origin.

There is reason to believe that the auditory powers of different individuals vary from one another within rather wide limits under conditions of health.

On this subject W. B. Dalby says:—"Amongst a very large number of people with the organs of hearing in an apparently healthy state, some few will be found upon whom throughout their lives certain notes produce no response. They will not for instance be able to hear the sounds made by grasshoppers, or the singing of some birds—the call of a partridge for instance; and in most persons, as age advances, the very high notes are lost. To prove this it is only necessary to blow one of Mr. Galton's whistles in a room full of people, when a considerable proportion of the assembly will fail to catch the high notes which are distinctly heard by the rest; and although this failure is also noticeable in many nervous affections, all other sounds will perhaps be heard quite normally by these individuals." Again, while musicians can detect the most minute differences of tone or pitch, there are many individuals whose hearing is otherwise good, whose 'musical ear' is altogether defective. Such individuals may not be able to distinguish one tune from another, and wrong notes do not jar upon their ear—not because they have not striven to educate their hearing in these directions, but because of some inherent nervous defect from which they suffer—and which may, roughly, be compared with colour-blindness as an analogous defect of the visual sense.

The hearing of blind persons is often præternaturally acute, because of the amount of attention which they are compelled to give to the impressions of this sense, and the increased power of discrimination that often follows as a consequence. An abnormal acuteness of hearing of a temporary character is, moreover, apt to supervene in some persons under certain conditions or states of the system to which reference will presently be made.

We cannot, in fact, so easily ascertain whether the hearing of a patient under examination is up to a normal standard of excellence, as we can ascertain whether he possesses a normal acuity of vision—nor do we, in fact, for diagnostic purposes take much note of slight deterioration in the patient's power of hearing. This is doubtless due in part to the fact that we cannot correlate slight or even grave impairments with visible changes in the terminal expansions of the auditory nerves, as we are able, in so many cases, to correlate visual defects with actual changes in the optic disc and retina.

As before stated, as a result of intra-cranial disease, defects of sight are, in a large proportion of the cases, double; on the other hand, as a result of intra-cranial disease, defects of hearing are, perhaps in a still larger proportion, single. Hence, in practice, we have oftentimes the sound ear as a foil, against which we may test the amount of impairment of hearing on the affected side—and an ever ready means of doing this is by ascertaining at what distance the ticking of a watch can be heard on the two sides. Of course this is only a rough relative test, watches differing so much in the loudness of their tick.

We must now pass briefly under review the various conditions under which deafness of primarily nervous origin is met with.

Deafness is sometimes congenital, and is then almost always bilateral. It entails dumbness in after life, when we have the condition known as deaf-mutism, often due to defective development of the internal or middle ear.

In the infinitely more frequent cases in which deafness shows itself first in after life, it may be more or less permanent, single or double, and due to structural or to functional causes.

In cases of primary nervous deafness due to organic disease the morbid process may exert its influence (*a*) upon the nuclei of origin and the root-fibres of the auditory nerves within the medulla; (*b*) upon the auditory nerves between the surface of the medulla and the internal auditory meatus; or (*c*) upon some portion of the auditory nerve contained within the temporal bone itself.

(*a*).—Disease of the nucleus of origin or of the root-fibres of the auditory nerve, on one side, may be caused by small focal lesions in the medulla (hæmorrhage or softening) or by tumours occupying the same situation. In these cases, the diagnosis would have to be made by other coexisting signs of disease of the medulla. Thus, with either of the pathological causes just mentioned, the contiguous nucleus and root-fibres of the facial would be very apt to be affected. In disseminated sclerosis, however, the nucleus or root-fibres of the auditory nerve may be involved on one, or even on both sides as I have seen, without any implication of the facial nerves.

(*b*).—The auditory nerves between the surface of the medulla and the internal meatus may become implicated by basal meningitis of a specific or non-specific character, their sheaths may be thickened or the seat of new growths, or they may be pressed upon by new growths originating in other parts, as in the under and inner part of the lateral lobe of the cerebellum or of its 'flocculus.' Again, deafness has been known to have been produced on one or both sides, in cases of incomplete thrombosis of the basilar artery (where death has not rapidly occurred), presumably owing to the fact that the minute vessels which supply the auditory nerves and the internal ear are given off from this trunk, and are therefore liable to be occluded at their point of origin.

(*c*).—Within the temporal bone itself the auditory nerve as well as its terminal expansions may also be injured in various ways, to such an extent as to produce deafness. Thus, a fracture of the skull, passing through the temporal bone, may tear across the auditory nerve fibres; or it may cause pressure (by extravasated blood) upon both the auditory and the facial nerves within the internal auditory canal; or, lastly, the fracture may produce deafness by damaging the cochlea. Sometimes deafness may follow severe blows or falls on the back of the head, where no fracture is produced, in a way that cannot be exactly defined. Exostoses, tumours of the temporal bone, syphilitic processes of various kinds, or ordinary degenerative changes, may also involve the nerve or the cochlea in such a way as to produce unilateral deafness. Most of the cases of deafness occurring in old age would belong to this latter category.

Under this head, we must also call attention to the deafness met with in 'Menière's disease.' In this affection paroxysms of vertigo are apt to occur from time to time, together with noises in the ears, possibly vomiting, and temporary deafness. The impairment of hearing on the side affected is apt to go on increasing with successive attacks—this affection being supposed to depend upon some morbid conditions of the semicircular canals. The vertigo and the sickness met with in Menière's disease are supposed to point to an implication of the portion of the eighth nerve that supplies the semicircular canals (the so-called 'space-nerve' of Cyon). Precise knowledge on this subject is, however, as yet wanting; though it is a direction in which further information is much needed, on the physiological and clinical as well as on the pathological side.

In reference to the deafness that so commonly supervenes in the subjects of congenital syphilis Dalby makes the following remarks:—"In these patients the hearing power begins to fail between five and fifteen years of age (very seldom later in life), and proceeds to a very great and often total deafness; the period between good hearing and the extreme point of deafness arrived at varying from a few weeks to several years. From this cause children sometimes become in the course of a month or six weeks totally deaf, but such rapidity is exceptional. Experience has shown how powerless treatment is to arrest the progress of this affection, so that attention should be confined to preventing its subjects from becoming dumb" (Quain's 'Dictionary of Medicine,' p. 422). This latter complication is almost certain to happen soon in neglected cases, especially those in which the deafness supervenes at any time between the fourth and the seventh years of age.

Primary nervous deafness of functional origin may be encountered in association with very various conditions.

It may be produced by different toxic agents such as quinine, salicyclate of soda, or more rarely, lead. In these cases the deafness is double, and usually temporary.

Violent emotional disturbances, as Dalby says, "play a very large part in the destruction or suspension of hearing, and this is especially observable in the case of women." He adds, "The same effect has been noticed with men who have been subjected to prolonged mental strain, in connection with literary work, or during commercial crises" (*loc. cit.*, p. 581).

Of this type also is the deafness that occurs sometimes from actual physical shocks, such as blows on the head and boxes on the ear; or that following upon loud explosions near the ear, which is not uncommon with artillerymen and naval men (Dalby).

After mumps, diphtheria, and some fevers, deafness may also be left, the precise cause of which is uncertain, but in which no changes are to be made out in the middle ear, and where there is no reason for supposing the existence of any definite lesion of the auditory nerves, or of their nuclei in the bulb.

Lastly, in functional hemianæsthesia, hysterical or other, as we have elsewhere indicated (p. 153), deafness of a partial character occurs on the side affected.

Exalted hearing, or auditory hyperæsthesia, is a not very common condition, but it is met with occasionally in some nervous or hysterical persons, and also to a notable extent in individuals who are hypnotised. In this condition there is an increased ability to discriminate different sounds, and not a mere hyperalgesia, or state in which the slightest sound produces a painful impression—the latter being a condition not uncommonly met with in highly nervous or debilitated persons when affected with almost any kind of illness, or in patients suffering from meningitis or other inflammatory brain affection. These conditions are probably dependent, for the most part, upon abnormal states of the auditory centres in the medulla, and, more rarely, upon unnatural states of the peripheral terminations of the nerve in the auditory apparatus itself.

A partial auditory hyperæsthesia, shown by an increased sensitiveness to high notes, may occur in certain cases of paralysis of the facial nerve, where the stapedius muscle is paralysed (p. 447), and a consequent increased tension of the tympanic membrane exists.

Perverted hearing. Under this head we may refer to the various cases (a) in which the patient is troubled with frequent or perhaps more or less constant subjective sounds or noises, and to others (b) in which the patient is troubled with auditory hallucinations.

(a).—What is known as tinnitus aurium, is the most common form of the subjective sounds with which patients are disturbed, and this may be referred to one ear only or to both. The precise nature of the sounds complained of varies much in different cases; they may be buzzing, humming, whistling, roaring, or clanging noises. These noises may last for longer or shorter periods, and then disappear for a time which is equally variable; or, in other distressing cases, the noises may be continuous.

Such subjective sounds or noises may be met with under very various conditions. They may occur, for instance, in association with different diseases of the external or middle ear. In other cases they are more distinctly of neuropathic origin, and then may owe their origin to diverse conditions. Thus, such noises may be brought on by the taking of large doses of quinine or salicyclate of soda; they may occur in debilitated subjects who are profoundly anæmic; or, they may be met with in association with various nervous affections, such as Menière's disease, epilepsy, the early stages of mania, or as sequences of sunstroke or malarial fever. In all these latter groups of cases tinnitus is apt to be an extremely intractable symptom. It may be met with, also, in cases of intra-cranial aneurysm. Finally, it may be an accompaniment of actual organic disease of different kinds, implicating either the auditory nerves or their nuclei in the medulla.

In persons who are overworked and the tone of whose nerve-system is distinctly lowered, loud explosive sounds may occur, associated with some dream, soon after falling asleep. This symptom has been met with at times, also, in some epileptics.

(b).—Auditory hallucinations may be present in delirium, and are extremely common in association with subacute or with chronic mania, in which patients frequently imagine that they hear voices constantly repeating some words, or bidding them do certain acts—hallucinations that are often of an extremely dangerous character. In other cases, patients may be haunted by musical sounds, vocal or instrumental, which they are wholly unable to dismiss from their consciousness.

Defects of this latter type are, in all probability, always due to some disordered function in the cortex cerebri, and have, therefore, from a regional point of view, a totally different import from that which attaches to tinnitus and other mere subjective noises.

The condition commonly though badly named 'word-deafness,' is not one of deafness at all. It is really a defect of apprehension in the auditory sphere, and is, as we have already intimated (p. 292), indicative of a lesion in or contiguous to the left upper temporal convolution.

G.—THE GLOSSOPHARYNGEAL NERVE.

The glossopharyngeal is in the main a sensory nerve, though it contains also a few motor fibres, in part derived from the roots of the spinal accessory. It is the special nerve of taste for the posterior third of the tongue and for the soft palate; and is the nerve of common sensibility for the same regions, as well as for the upper part of the pharynx, the eustachian tube, and the tympanum. It supplies the following muscles:—the middle constrictor of the pharynx, the stylopharyngeus, the levator palati and the azygos uvulæ.

In accordance with facts and views already referred to (p. 447), a part of the nucleus of the glossopharyngeal also ministers to the sense of taste for the anterior half or two-thirds of the tongue, through the intermediation of the chorda tympani and the nerve of Wrisberg.

This nerve is one that is rarely or ever paralysed alone. It pursues a deep course to its termination, and is, in this respect, wholly unlike either the facial or the trigeminal nerves which, in their longer and more exposed course towards the periphery, are liable to become implicated in many different kinds of morbid processes.

The roots of the glossopharyngeal nerve or its nucleus may, of course, be involved by disease situated in the bulb, but in that case it is extremely likely to be affected in concert

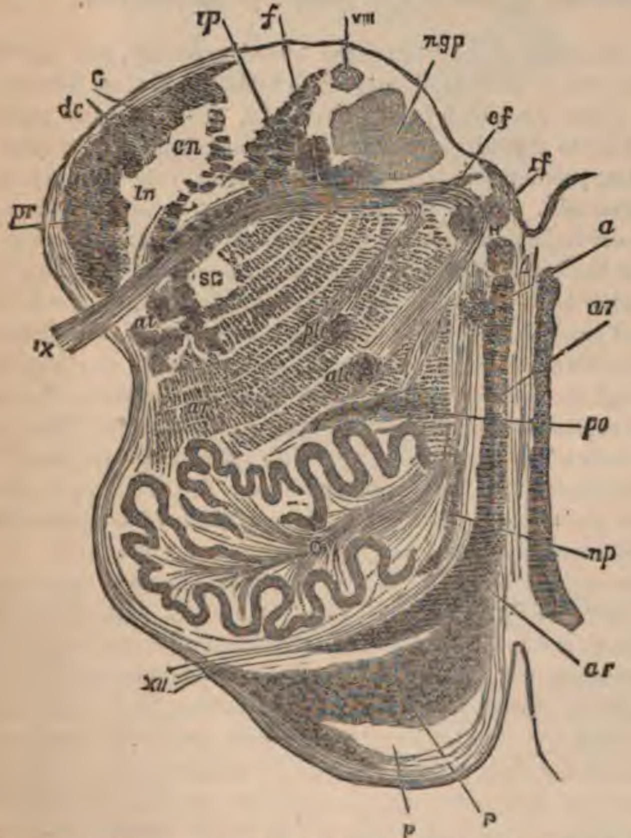

FIG. 106.—SECTION THROUGH THE BULB ON A LEVEL WITH THE GLOSSOPHARYNGEAL NERVE [after Young and Ross].

P, The anterior pyramid; *p*, its accessory or latest developed portion.
xii, Hypoglossal nerve. H, Nucleus of the hypoglossal (the internal, antero-lateral, and postero-lateral groups of cells); *a*, anterior group of cells.
ix, Glossopharyngeal nerve. *ngp*, Nucleus of the glossopharyngeal nerve.
viii, Lower part of the posterior median auditory nucleus.

if, Internal accessory facial nucleus.
ef, External accessory facial nucleus.
alc, Anterior nucleus of the lateral column. *plc*, Posterior nucleus of the lateral column.
f, Fasciculus rotundus.
ip, Internal division of the inferior peduncle of the Cerebellum. G, Column of Goll. *dc*, Direct cerebellar tract lying on the surface of the posterior root zone (*pr*), and the ascending root of the trigeminus (*at*).
cn, Clavate nucleus. *tn*, Triangular nucleus. SG, Substantia gelatinosa. L, Posterior longitudinal fasciculus.
ar, Portion of the formatio reticularis representing the internal division of the anterior root-zone of the spinal cord. *ar′*, Do. representing the external division of the anterior root-zone of the spinal cord.
O, Olivary body. *np*, Nucleus of the pyramid. *po*, Parolivary body.

with the auditory, the vagus, the spinal accessory, and the hypoglossal (one or more of them), whose nuclei are in close relation with that of the glossopharyngeal (Figs. 79, 106). In this region we may have to do with softening, small hæmorrhages, infiltrating new growths, patches of sclerosis, or slow degenerative changes in the ganglion cells. The upper part of the spinal accessory nucleus, and the lower facial nucleus, first described by L. Clarke, together with that of the hypoglossal nerve, are particularly prone to be involved in this latter kind of change, with the result of producing a well known form of paralysis (labio-glosso-laryngeal paralysis). In this disease the sensory fibres of the glossopharyngeal are rarely or ever implicated, although the muscles supplied by its reputed motor fibres often are—a fact explicable enough if, as we suppose, these motor fibres really take their origin from the nucleus of the spinal accessory, though they are distributed with the glossopharyngeal. This being so, the nucleus of the glossopharyngeal nerve would not be one of the parts affected in labio-glosso-laryngeal paralysis.

One mode, however, in which the glossopharyngeal nerve may be implicated together with the vagus, the upper part of the spinal accessory, and the hypoglossal, is their implication at the surface of the medulla (Fig. 107) by processes of chronic meningitis, by syphilitic thickenings, or by tumours of various kinds. The glossopharyngeal and some other of these nerves may likewise become involved during their passage through the occipital bone, owing to the existence of sarcomatous or other tumours affecting this part, or to the presence of some other form of disease that narrows the foramina through which they pass out of the cranial cavity.

The diagnosis of paralysis in the sphere of the glossopharyngeal nerve must always practically rest upon the implication of its sensory fibres—that

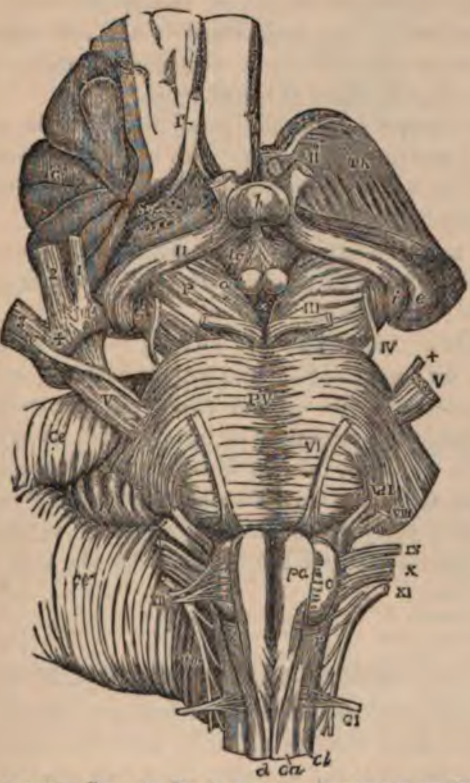

FIG. 107. PARTS ABOUT BASE OF BRAIN SHOWING RELATIONS OF CRANIAL NERVES [after Allen Thomson].

On the right side the convolutions of the Island of Reil have been left, together with a small part of the anterior cerebral convolutions. On the left side these have been removed by an incision carried between the Thalamus and the Cerebral Hemisphere.

1', The Olfactory tract cut short and lying in its groove. II, Left Optic Nerve in front of the commissure. II, The right optic tract. Th, The cut surface of the Left Thalamus. C', Island of Reil. Sy, Fissure of Sylvius. *xx*, Anterior perforated space. *e* and *i*, The external and internal corpus geniculatum, respectively.

h, Pituitary body. *tc*, Tuber cinereum with the Infundibulum. *a*, One of Corpora albicantia. P, Crus cerebri. *f*, The fillet. III, Third nerve. *x*, The posterior perforated space.

PV, Pons Varolii. V, The greater root of the fifth nerve. *x*, The lesser or motor root. VI, Sixth nerve. VII, The facial. VIII, The auditory. IX, The glosso-pharyngeal. X, The pneumo-gastric. XI, The spinal accessory nerve. XII, The hypoglossal.

CI, The suboccipital or first cervical nerve. *pa*, Pyramid. *o*, Olive. *d*, Anterior median fissure of the cord, above which the decussation of the pyramids is represented. *ca*, Anterior column of cord. *r*, Lateral tract of medulla continuous with *cl*, the lateral column of the spinal cord.

is upon the impairment or loss of the special sense of taste in the posterior third of the tongue and in the soft palate, and also upon the impairment or loss of common sensibility in the same parts and in the upper part of the pharynx.

When the disease causing the paralysis affects the nucleus of the nerve, and involves that part with which the nerve of Wrisberg is connected (p. 447), we may also expect to meet with loss of taste in the anterior half of the tongue.

We may, as a rule, expect to find paralysis of the glosso-pharyngeal nerve associated with paralysis of some one or more of the cranial nerves above referred to.

Irritative lesions of the glosso-pharyngeal nerve are far from common, and little is definitely known as to the effects of irritation of its sensory fibres. Theoretically, it ought to give rise to subjective sensations of taste, as well as hyperæsthesia in the regions in which the nerve is distributed. Spasms of the muscles innervated by the motor fibres distributed with the glosso-pharyngeal are much more likely to be due to centric than to peripheral irritation, and will, therefore, be referred to under the head of the spinal accessory nerve.

H.—THE PNEUMO-GASTRIC OR VAGUS NERVE.

The pneumo-gastric is the great cerebral systemic nerve. Its area of distribution is far wider than that of any other cranial nerve, since it supplies branches to the pharynx, the larynx, the lungs, the heart, the œsophagus, the stomach, the intestines, the liver, the spleen, and possibly to the kidneys and internal organs of generation.

Its fibres are in the main sensory, though motor fibres are bound up with them, derived principally from the spinal accessory.

As may be seen by reference to Fig. 108, the fibres of the pneumo-gastric take their origin in the medulla from a grey nucleus, near the calamus scriptorius, situated immediately above that of the spinal accessory and below that of the glosso-pharyngeal nerve, having on its inner side the hypoglossal, and on its outer side the inferior trigeminal nucleus.

The fibres of the pneumo-gastric issue from the side of the medulla immediately below those of the glosso-pharyngeal (Fig. 107). After passing out through the jugular foramen, behind the glosso-pharyngeal nerve and in the same sheath with the spinal accessory, the vagus

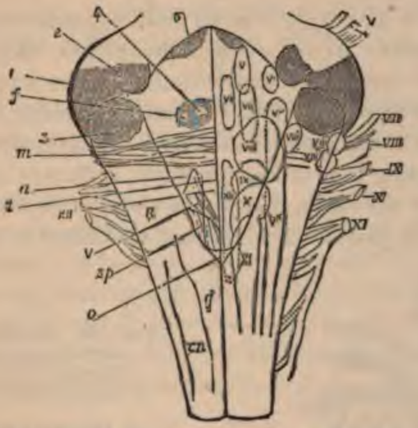

FIG. 108. DIAGRAMMATIC VIEW OF THE POSTERIOR SURFACE OF THE MEDULLA, OR FLOOR OF THE FOURTH VENTRICLE, SHOWING THE RELATIVE POSITIONS OF DIFFERENT NERVE NUCLEI [after Erb].

The left half of the figure represents :—Cn, funiculus cuneatus, and *g*, funiculus gracilis; O, Obex; *sp*, nucleus of the spinal accessory; *p*, nucleus of the pneumogastric; R, restiform body; XII′ nucleus of the hypoglossal; *t*, funiculus teres; *a*, nucleus of the acousticus; *m*, striæ medullares; 1, 2, 3, middle, superior and inferior cerebellar peduncles. *f*, fovea anterior; 4, eminentia teres (genu nervi facialis); 5, locus cœruleus.

The right half of the figure represents the nerve nuclei diagrammatically :—V, motor trigeminal nucleus; V′, median, and V″, inferior sensory trigeminal nuclei; VI, nucleus of abducens; VII, facial nucleus; VIII, posterior median acoustic nucleus; VIII′, anterior median; VIII″, posterior lateral; VIII‴, anterior lateral acoustic nuclei; IX, glosso-pharyngeal nucleus; X, XI, XII, nuclei of vagus, spinal accessory, and hypoglossal nerves respectively. The Roman numerals at the side of the figure from V to XI represent the corresponding nerve roots.

swells into the large 'ganglion of the trunk'; and after receiving the internal division of the spinal accessory, it passes down the neck behind and in the same sheath with the carotid arteries. The nerve of the right side enters the thorax over the subclavian artery, and thence passes along the side of the trachea to the back of the root of the lung, where it spreads out into the posterior pulmonary plexus; from this point it passes down by the side of the œsophagus, and thence to the posterior surface of the stomach and the solar plexus. The nerve of the left side enters the thorax between the subclavian and the carotid arteries; then passes behind the left innominate vein and over the arch of the aorta, to the back of the root of the right lung, where, after spreading out and joining with its fellow, it proceeds as a single cord, along the front of the œsophagus to the anterior surface of the stomach, and thence on to join the left hepatic plexus.

Before speaking of the lesions or accidents that may affect the trunk of the nerve, it will be most expedient to deal with the subject of lesions of its several branches, indicating, that is, the effects of destructive and irritative lesions upon the functions of the parts or organs to which they are distributed.

In affections of the trunk of the nerve, the exemption of this or that branch, proceeding from above downwards, suffices to enable us to diagnose the site of the lesion, that is, the situation in the trunk at which the lesion exists, just as we do in cases of disease of the facial nerve.

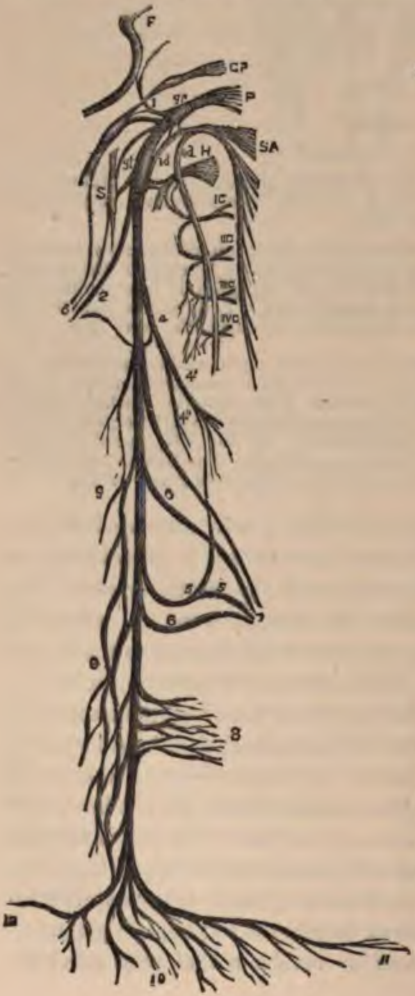

FIG. 109. DIAGRAM OF THE PNEUMOGASTRIC AND SPINAL ACCESSORY NERVES, THEIR CONNECTIONS AND BRANCHES [after Hermann].

P, Pneumo-gastric nerve.
SA, Spinal accessory nerve.
H, Hypoglossal nerve.
GP, Glosso-pharyngeal nerve.
F, Facial nerve.
S, Superior cervical ganglion of the Sympathetic.
gr, ganglion of the root of the vagus.
gt, ganglion of the trunk of the vagus.
1, Auricular branch of the vagus (Arnold's nerve).
2, Pharyngeal branch.
3, Convergence of nerves to form pharyngeal plexus.
4, Superior laryngeal nerve.
  4', Internal branch of superior laryngeal nerve.
  4'', External branch of superior laryngeal nerve.
6, 6, 6, Cardiac branches of the vagus.
5, Inferior or recurrent laryngeal nerve.
  5, Cardiac branch of inferior laryngeal nerve.
7, Convergence of branches of vagus to form cardiac plexuses.
8, Pulmonary branches.
9, Œsophageal branches.
10, Gastric branches.
11, Splenic branches.
12, Hepatic branches.
SA, Spinal accessory nerve.
id, Internal division of spinal accessory.
ed, External division of spinal accessory.
IC, IIC, IIIC, IVC, Cervical nerves.

Hence the importance of remembering the order and different levels at which the several branches are given off from the parent trunk (Fig. 109).

The **auricular branch** of the vagus arises from the ganglion of the root and is the common sensory nerve for the integument of the back of the pinna.

The **pharyngeal branch** is composed in the main of motor fibres derived from the spinal accessory. It comes off from the vagus opposite the lower part of its large ganglion, and combines with branches from the glosso-pharyngeal and the sympathetic to form the pharyngeal plexus, which innervates the pharynx and the soft palate. These parts may be paralysed separately, but are more frequently affected in combination, either bilaterally or unilaterally.

THE PALATE.

It will be most convenient to refer first to paralysis of the palate. The opinion, formerly entertained, that one of the most important muscles of the palate, the levator palati, was innervated by the facial nerve, and that paralysis of this muscle is one of the signs of a lesion of the trunk of the facial on the proximal side of the geniculate ganglion has been already referred to and its inaccuracy pointed out (p. 446). It is now beginning to be more commonly recognized that this levator palati, like other of the palate muscles, is, in all probability, innervated by the spinal accessory, through the pharyngeal branch of the vagus.

When **paralysed on both sides** the palate hangs low and is flaccid. It is not raised during breathing or in phonation ; the common mode of detecting this latter defect being, to make the patient utter the vowel sound 'ah' in a high tone, while the observer watches the palate, to see whether or not it is raised (as it should be) during the utterance of this sound. Touching the surface of the palate itself, moreover, causes no reflex movement. Deglutition is interfered with, as the soft palate cannot be raised so as to shut off the posterior nares. For the same reason, the patient's speech becomes nasal in character, assuming a characteristic 'twang'; and difficulty is experienced in properly pronouncing the explosive consonants *p* and *b*—which, with such patients, tend to become *f* and *v*.

In **unilateral paralysis** of the palate, the signs are altogether less marked. On inspection the paralysed side of the palate is seen to be lower and less arched than its fellow ; when the patient is made to utter the sound 'ah,' the immobility of the paralysed side contrasts with the elevation and lateral deviation of the palate on the sound side; again, touching the palate on the paralysed side with the end of a quill pen or pen-holder causes no reflex movement, such as may be evoked by a similar touch on the non-paralysed side. The position of the uvula is a sign that is not to be depended upon, since this is subject to variation in different individuals independently of paralysis. Unilateral paralysis of the palate causes little or no interference with deglutition; articulation, also, may not be interfered with at all, or, at most, the voice may reveal a slight nasal 'twang.'

THE PHARYNX.

The muscles of the pharynx are probably innervated almost exclusively by fibres ultimately derived from the spinal accessory nerve though distributed with the pharyngeal branch of the vagus. Paralysis of the muscles of the pharynx like those of the palate may be bilateral or unilateral. Where the **paralysis is unilateral** the patient's power of swallowing may be interfered with although not gravely impaired. **Bilateral paralysis** of the pharyngeal muscles, however, causes extreme difficulty in swallowing. Solids are apt to lodge at the back of the tongue, between it and the epiglottis, or over the latter, causing great dyspnœa and threatening suffocation; fluids are apt to run into the larynx, producing symptoms of suffocation and violent paroxysms of coughing.

Where the paralysis is limited to the superior constrictor of the pharynx, fluids tend to regurgitate through the nose during deglutition, even where there is no paralysis of the palate. This regurgitation becomes much more marked, where, as is very often the case, such partial paralysis of the pharynx is associated with paralysis of the soft palate.

There are only two conditions causing difficulty of deglutition that are at all likely to be confounded with paralysis of the muscles of the pharynx. The first of these is organic disease of the lower end of the pharynx, in the form of a carcinomatous or other kind of new growth. But where the history of the case leaves any room for doubt on this head, it may be easily set at rest by the passage of a bougie. Precisely the same thing may be said in reference to the second of the two conditions above referred to, viz., spasm of the pharyngeal muscle. Of course spasm may, and often does, coexist with organic disease. In other cases, it may be caused by irritating rather than paralysing lesions of the nerve fibres supplying these muscles. Far more frequently, however, it occurs as a pure neurosis. Then, it may be met with separately in simply nervous subjects; or it may occur in association with other spasmodic symptoms, in hysteria, or in hydrophobia.

The problems of regional and pathological diagnosis in cases of paralysis of the pharynx are usually very closely related to one another. In the first place, it may be said that this form of paralysis is very rarely caused by disease outside the cranium; as the pharyngeal branch is given off very high up in the neck, it is not liable to be involved in those cases of paralysis of the vagus due to various pathological states or injuries implicating the trunk of the nerve in different parts of the neck. It is met with especially in cases (*a*) of disease of the medulla or of the pons Varolii, when it is generally associated with more or less paralysis of the tongue, and it may be of the lips and face. In apoplectic states, as well as in later stages of disease due to hæmorrhage into, or softening of, the pons, dysphagia is commonly a very marked symptom. It may also be associated with tumours in this situation or in the medulla oblongata. Again it is one of the components of various bulbar paralyses, to whatever pathological conditions they may be due (p. 355). In another group of cases, paralysis of the pharyngeal muscles is due (*b*) to disease implicating the upper roots of the spinal accessory at the surface of the medulla. Among such causes we may mention chronic meningitis, tumours, syphilitic thickenings of the sheaths of the nerves, or compression by an aneurysm of the vertebral artery. The paralysis of this type that follows diphtheria is commonly supposed to be due to some morbid condition of the roots of the nerves, mostly of a transitory character. In this latter case we generally have to do with a bilateral paralysis; but in the case of local organic disease at the surface of the medulla, the paralysis of the pharynx is frequently unilateral, and in such cases it may, as Hughlings Jackson has observed, be combined with paralysis of the palate as well as with that of the vocal cord and the half of the tongue on the same side. In a final group of cases (*c*) paralysis of the palate and pharynx (mostly unilateral) may be caused by some disease of the occipital bone involving the spinal accessory nerve as it passes through the jugular foramen. In such a case, there would be a strong chance of a coincident implication, more or less grave, of the trunk of the pneumo-gastric (contained within the same sheath), and likewise of the glosso-pharyngeal which passes with the other two nerves through the same foramen.

Paralysis of the **branches** of the vagus **to the œsophagus** and **to the stomach** rarely or ever occurs alone (that is, as a result of disease of these particular branches themselves), though such paralyses are met with, from time to time, as a consequence of disease or injury to the trunk of the nerve itself.

THE ŒSOPHAGUS.

Paralysis of the œsophagus is all the less likely to be met with, except as a condition of great rarity, because, for its production to a marked degree, paralysis of the nerves of both sides would be needed. When such bilateral paralysis of the œsophagus exists, solid food would have a tendency to lodge principally in the upper or lower part of the tube. Fluids and some portions of solid food would, however, if once passed from the pharynx into the œsophagus, descend to some extent into the stomach by mere gravitation. Organic disease of the œsophagus itself, or the narrowing of the tube by the pressure of some morbid product acting from without, would similarly tend to impede the passage of food through the tube; but such causes of dysphagia may be distinguished, if need be, from paralysis of the œsophagus, by the great ease with which a sound can be passed into the stomach in the latter case.

THE STOMACH.

Paralysis of the branches of the pneumo-gastric supplied to the stomach leads to two distinct classes of symptoms, because the principal branches supplied to this organ are sensory, though others of them (really derived from the spinal accessory), have motor functions.

The feeling of hunger is usually supposed to be a sensation dependent in the main upon impressions reaching the brain through the sensory fibres of the vagus, although evidence derivable from disease of this nerve by no means points exclusively in this direction. In one case, recorded by Dr. Geo. Johnson, in which there was softening of the medulla and pressure upon the roots of origin of the left pneumo-gastric by an aneurysm of the vertebral artery, there was a complete loss of the feelings of hunger and thirst. The same kind of defect was met with also by Grasset in a case of labio-glosso-laryngeal paralysis. Appetite is, however, not always lost in animals in whom the pneumo-gastrics have been cut; quite the reverse state of things has sometimes existed. The appetite of some animals has seemed to be insatiable, and they have devoured enormous quantities of food. In two cases of disease of both pneumo-gastrics, occurring in man, the same insatiable appetite was noticed. In one, this symptom coexisted with dyspnœa, and vomiting of unaltered food, and after death both pneumo-gastrics were found to be atrophied (Swan); in the other case, both pneumo-gastrics were the seat of small neuromata. This tendency to eat enormously is probably to be expected in most animals in the absence of a feeling of satiety. The presence of food may cause such animals to go on eating, in cases where this customary check is absent; whilst for them, as well as for those human beings in whom as a consequence of disease of the pneumogastrics the feeling of satiety has been absent, there may be, in addition, some uneasy sensation felt at the epigastrium, acting as a positive factor, and leading to the taking of food for its relief—a kind of false hunger, in fact, accompanied by the absence of a feeling of satiety.

The pneumo-gastric is also, in part, the motor nerve of the stomach so that, when both nerves are paralysed there would be a diminution in the contractile power of the organ; and, on the other hand, when they are irritated, vomiting may occur. The vomiting that occurs through the intermediation of the vagus may be due to peripheral irritation (when it is certainly of reflex origin); to irritation of some part of the trunk of the nerve; or to irritation of the centres of the pneumo-gastric or spinal accessory nerves in the medulla. In the latter cases it may be sometimes a reflex phenomenon, and sometimes a direct result of irritation of the motor fibres going to the stomach with the pneumogastric. Probably the mechanism is generally reflex when the trunk of the nerve is irritated by the growth of some tumour. Boinet, having exposed one of the vagus nerves during the progress of an operation in the neck, noticed that, whenever he touched the nerve, the patient vomited.

Concerning other effects resulting from irritation of the branches of the vagus supplied to the stomach, pathology supplies us with no definite information. Of the different neuroses referable to a disordered action in the nerves of the stomach, it is, moreover, difficult to say anything definite from pathological evidence. We stand in need of more exact knowledge concerning the part taken in gastralgia, pyrosis, bulimia, polydipsia, by the stomachal branches of the vagus and the sympathetic respectively. These are all neuroses of uncertain origin, though there is much probability that a perverted activity of the vagi and their medullary centres is mainly concerned with the production of the first of them.

INTESTINES, LIVER, AND OTHER ABDOMINAL ORGANS.

Pathology and clinical observation have as yet furnished us with no definite information concerning the effects of paralysis or of irritation of the branches of the vagus distributed to the above mentioned organs. We are met here with the difficulty referred to in connection with the stomach, and perhaps, in a still greater degree, of distinguishing between the morbid symptoms that should be referred to the vagus and to the sympathetic respectively; and, likewise with the further difficulty of discriminating those which are caused by paralysis from those due to irritation of the branches of either of them.

LARYNX.

Of the two branches of the vagus supplied to the larynx, the superior laryngeal, which comes off from the trunk of the nerve at about the level of the third cervical vertebral, is an exclusively sensory nerve, except that it supplies the crico-thyroid muscle, which is the tensor of the vocal cords. The inferior or recurrent laryngeal is, however, purely motor, and supplies all the other muscles of the larynx. The motor fibres of both these nerves, proceed really from the nucleus and roots of the spinal accessory. The right recurrent nerve separates from the vagus in the lower part of the neck opposite the subclavian artery, thence it winds beneath and behind this vessel on its way upwards to the larynx; whilst the left recurrent laryngeal winds round the arch of the aorta and ascends beneath the common carotid to the larynx.

Paralysis of the motor fibres of the superior laryngeal, supplying the crico-thyroid muscle, would give rise to no very appreciable results, when unilateral; though the result of the bilateral paralysis of this nerve, would be to render the voice hoarse and deep, and to make the production of high notes more or less impossible, owing to defective tension of the vocal cords.

Paralysis of the sensory fibres of the superior laryngeal rarely occurs alone, but usually as part of the symptoms caused by an affection of the trunk, the roots of origin, or the nucleus of the vagus. In the latter case, the loss of sensibility may be bilateral; but in the former it is much more likely to be unilateral, and is then not nearly so dangerous a condition, since the contact of food or of any foreign body with the sensitive side of the larynx will still excite a violent expulsive fit of coughing.

Where these sensory nerve fibres are irritated, or where their centres are in a condition of increased irritability from any cause, we have a condition of hyperæsthesia rather than of anæsthesia, the existence of which is apt to lead to attacks of spasmodic coughing, such as may be met with at times in nervous excitable persons. In other cases there may be, in such persons, perverted sensations (paræsthesiæ) referred to the larynx, causing them to believe that a foreign body of some kind is lodged therein.

The effects of paralysis of the motor fibres of the superior and of the recurrent laryngeal, supplying the muscles of the larynx, would be found to be a very complicated subject, were it to be followed out in all its details. This will not be necessary in such a work as the present, since it is the paralysis of the abductors and of the adductors of the vocal cords which is of by far the most consequence; that is of the posterior crico-arytenoideus (the chief abductor), together with the lateral crico-arytenoideus and the arytenoideus (the two adductors), all of them being muscles supplied by the recurrent laryngeal nerves.

A few preliminary words, by way of explanation, together with the following diagrams, may be desirable for the better understanding of this subject.

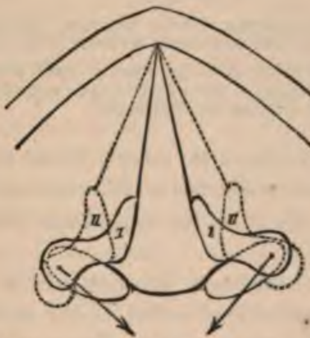

FIG. 110. SCHEMA OF A HORIZONTAL SECTION THROUGH THE LARYNX, SHOWING THE POSITION OF THE VOCAL CORDS DURING QUIET BREATHING [after Landois].

1,1, The position of the arytenoid cartilages and vocal cords during quiet breathing. The arrows indicate the direction in which the posterior crico-arytenoid muscles act (the chief abductors). 11, 11, Position of the arytenoid cartilages in consequence of the contraction of these muscles (that is position during deep breathing).

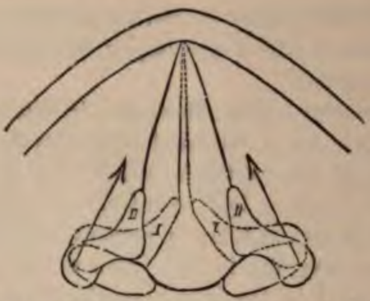

FIG. 111. SCHEMA OF A HORIZONTAL SECTION THROUGH THE LARYNX, ILLUSTRATING THE ACTION OF THE LATERAL CRICO-ARYTENOID MUSCLES [after Landois].

11, 11, Position of the arytenoid cartilages during quiet breathing. The arrows show the direction in which the lateral crico-arytenoid muscles act. 1, 1, Position of the cartilages when the muscles are contracted.

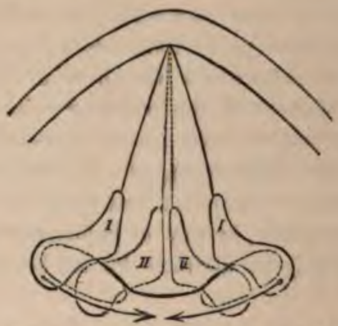

FIG. 112. SCHEMA OF A HORIZONTAL SECTION THROUGH THE LARYNX, ILLUSTRATING THE ACTION OF THE ARYTENOID MUSCLES [after Landois].

1, 1, Position of the arytenoid cartilages during quiet breathing. The arrows show the direction of the action of the arytenoid muscles. 11, 11, Position of the cartilages when the muscles are contracted.

The glottis assumes the most various forms under different circumstances, owing to the existence of different degrees of divergence of the vocal cords. Four of these positions may be specified as follows :—

(1).—Complete closure of the glottis (Fig. 112, II, II) such as is produced by the combined action of the arytenoid and of the lateral crico-arytenoid muscles. (2).—The cadaveric position, being the position in which the vocal cords are found after death, that is slightly separated from one another. (3).—The position of quiet breathing in which the glottis is more open than in the last position (Fig. 110, I, I), though still only moderately dilated. (4).—The position of deep inspiration, in which the glottis is widely dilated (Fig. 110, II. II).

As **unilateral paralysis of the laryngeal muscles** is decidedly more common than bilateral paralysis, we shall first speak of the signs and symptoms to which it gives rise. Soon after the initiation of such a paralysis, it may be seen, on laryngoscopic examination, that the vocal cord on the affected side remains persistently in a position of slight abduction, that is, it is nearer the middle line than it would be in healthy quiet breathing and has fallen into the so-called 'cadaveric position.' It cannot be brought to the middle line as the other cord is during phonation, nor can it be moved outwards like its fellow during deep inspiration. The voice may be hoarse or it may be but little altered, as in some cases the healthy cord can be brought even a little beyond the middle line, and consequently so near to the paralysed cord as to give rise only to a very slight defect in phonation. Attempts at coughing are very ineffective, which is easily to be understood, seeing that complete approximation of the vocal cords is impossible, and, therefore, the preliminary step essential for the occurrence of an explosive cough must be absent.

Sometimes in cases of complete unilateral paralysis, the vocal cord on the affected side is found, not to be in a state of semi-abduction, but to be brought almost up to the middle line. This is probably to be looked for in all cases in which the paralysis has existed for some months, and may be explained by a kind of tonic contracture occurring in the, now unopposed, crico-thyroid muscle of the same side (supplied by the superior laryngeal), the effect of which would be to over-extend the vocal cord, and consequently to bring it nearer to the middle line (Poore). Where, with unilateral paralysis, the cord is in this position, there is usually no alteration of the voice, though it may be slightly high pitched. On laryngoscopic examination the cord is seen to remain motionless in inspiration—not moving outwards as the cord on the healthy side does.

Where unilateral paralysis of the laryngeal muscles is caused by an affection of the root fibres of the spinal accessory at the surface of the medulla, it has been observed that the affected vocal cord remains, throughout, in the semi-abducted position (Gowers), which is only what might have been expected, seeing that in such a case the crico-thyroid supplied by the superior laryngeal would be paralysed as well as the muscles supplied by the recurrent laryngeal nerve.

Bilateral paralysis of the muscles supplied by the recurrent laryngeal nerves is decidedly less common than the unilateral paralysis. When it occurs there may be the same kind of difference as to the position of the vocal cords; that is, in the early stages, they may both of them be in the 'cadaveric position;' while in the later stages they may be in a position of more marked adduction, owing to contracture of both crico-thyroid muscles. When they are in the first of these positions, as the loss of adduction during phonation is the most prominent defect, the condition is apt to be described merely as 'paralysis of the adductors;' on the other hand, when they are in the second of these positions, the loss of abduction during inspiration being now the most noticeable defect, the condition is apt to be spoken of merely as 'paralysis of the abductors.' This, however, is a partial and faulty description; since in each case the vocal cords are really motionless in either direction.

The difference between the symptoms associated with these two positions of the vocal cords is very marked. Thus, when the vocal cords are in the position of **semi-abduction,** phonation is more or less impossible, and the power of coughing is similarly lost. On the other hand, whilst the patient is quiet, or so long as he does not exert himself overmuch, there is no appreciable dyspnœa. When, however, the paralysed cords are in the position of **adduction,** the voice is not nearly so much interfered with, though his respiratory power is gravely impaired. Phonation is comparatively easy, though the voice is high pitched and stridulous. The patient's power of coughing is very imperfect, because although the cords are close to one another, they cannot be further approximated so as completely to close the glottis. Again, the inability to separate the vocal cords during inspiration, only permits the patient to breathe easily so long as he is at rest, and his respiration is not hurried. Even a slight amount of exertion may speedily produce marked dyspnœa with stridulous inspiration.

In addition to these graver forms of actual paralysis, concerning the causation of which I shall presently speak, mention must be made here of the mere **paresis** (mostly of functional origin) which, under certain circumstances, shows itself in some of the muscles of the larynx—especially in the adductors, giving rise to a loss of voice. An aphonia of this type is met with often in persons suffering from extreme weakness or prostration, from whatever cause arising; or after excessive use of the voice in speaking, singing, or screaming, in persons not so delicate; also, in hysteria, or from inflammation in the neighbourhood of the vocal cords, either above or below—as when a patient affected with an attack of bronchitis loses his voice. These are mere temporary defects, for the most part; and even while they last, it is of interest to note that the power of adduction is merely lost under the influence of cerebral speech incitations, since such patients are perfectly well able to cough, showing that, in each of the above-mentioned conditions, perfect adduction can still be brought about under the influence of any strong reflex stimulus capable of inciting the act of coughing.

We must now turn to the question of causation, or, in other words, to the problems connected with a *pathological diagnosis* in cases of unilateral and bilateral paralysis of the muscles of the larynx.

As already stated, **unilateral paralysis of the laryngeal muscles** is more common than the bilateral affection, and it is commonly due to an implication of one of the recurrent nerves by some injury or morbid process. Thus, one of these nerves may be damaged by some gunshot wound or stab, or by some surgical operation in the neck; or it may be compressed by enlarged glands, or tumours of various kinds, or by aneurysms of the carotid and subclavian arteries or even of the aorta itself. Within the thorax, either of these nerves may be involved and compressed by mediastinal tumours, or by pleuritic adhesions about the apex of the lung.

Morbid conditions that involve the trunk of the vagus itself, before the giving off of the recurrent laryngeal nerve, may also cause unilateral paralysis of the laryngeal muscles; while, if the disease should likewise be situated above the origin of the superior laryngeal nerve, there may be, in addition to the paralysis of the crico-thyroid muscle, some loss of sensibility in and about the affected side of the larynx.

When situated above the ganglion of the trunk (Fig. 109, *gt*), disease of the vagus no longer causes paralysis of the laryngeal muscles. From this level upwards, for the production of any such result it is the accessory branch of the spinal accessory, or the roots or nucleus of origin of this branch in the medulla, that must be affected, in order to produce paralysis of the laryngeal muscles. The common causes of such implication of the spinal accessory roots and nucleus have been already referred to (p. 488); it is only necessary to state here that in such forms of disease, the crico-thyroid muscle may be paralysed together with the muscles supplied by the recurrent laryngeal. This occurrence has the effect of preventing the late supervention of the position of adduction by the paralysed cord (p. 493).

Bilateral paralysis of the laryngeal muscles is most commonly met with where the nuclei of origin, in the medulla, of the accessory branches of the spinal accessory nerves are diseased; where laryngeal paralysis occurs as a sequence of diphtheria; and also in cases of mediastinal growths, in which both recurrent laryngeal nerves are involved. In almost all the other cases, above referred to, the paralysis is far more likely to be unilateral. Still, two remarkable cases have been recorded (Baümler, Johnson) in which, with pressure on one recurrent laryngeal and vagus, there was paralysis of both vocal cords (in the one equally, and in the other case unequally, the paralysis in the latter being less on the side opposite to, than on the side of, the compressed nerve). The cause of this is obscure, but Johnson suggests that the paralysis of the opposite side may be a kind of inhibitory paralysis, caused by pressure upon some of the afferent fibres of the vagus.

The double affection may also follow, occasionally, as a sequence of some acute febrile illness other than diphtheria (which has been already mentioned); or it may supervene in some cases of chronic poisoning by lead or arsenic.

The so-called rheumatic paralysis of the vocal cords, from exposure to currents of cold air, is commonly unilateral; but it may be a bilateral affection.

Spasm of the laryngeal muscles rarely proceeds from irritation by tumours or other pathological conditions of the trunk of the vagus or even of the recurrent nerve. It is almost invariably a result (*a*) of some local irritation of the peripheral extremities of certain afferent nerves; or else (*b*) of some exalted irritability of certain nerve centres in the medulla.

When spasm of the laryngeal muscles is excited, it results invariably in a spasmodic closure of the glottis—owing to the fact that the adductors are far more powerful than the abductors of the vocal cords.

(*a*).—In some of the cases belonging to the first category, in which we have to do with spasms of reflex origin, the contractions are merely momentary; though they may recur at short intervals, as in cases in which spasmodic cough is excited, either by the breathing of irritating gases or by the contact of solids or liquids with the upper part of the larynx. Spasmodic cough may also be excited by irritation of other peripheral branches of the vagus, viz., those supplied to the throat, to the stomach, and occasionally even those of the branch supplied to the external auditory meatus. In children or in nervous subjects, in whom the reflex excitability is high, these manifestations are most apt to occur; and the same may be said concerning a more severe and durable form of spasm which is prone to manifest itself, especially at night, in children suffering from some catarrhal inflammation contiguous to the larynx.

(*b*).—In this second category are included all the cases in which spasm of the glottis arises from centric causes, mostly as component phenomena of some neurosis, affecting the nuclei of the accessory branch of the spinal accessory. It may thus be associated with hysterical convulsions, with eclampsia, with epilepsy, and with hydrophobia. Again, in whooping cough, spasm, with subsequent imperfect relaxation of the glottis, accounts for the peculiar nature of the cough. On the other hand, spasm of the glottis plays a much more prominent part in another neurosis of epileptic type, viz., laryngismus stridulus (also known as 'spasmodic croup' or 'child crowing').

Laryngismus stridulus is met with principally in infants or children under three years of age, though it also occurs more rarely in older children or even in adults. As with the more common manifestations of epilepsy, so here, the attacks commence quite abruptly. The child is roused, perhaps, from its sleep, or checked during the act of sucking, by a sudden catch in its breathing, more or less complete.

One of the earlier writers on this spasmodic affection, Dr. Ley, describes the attacks in the following terms:—

> "When the closure of the chink of the glottis is not perfect, the child struggles for its breath; the respiration is hurried; the countenance generally bluish or livid; the eyes staring; and each inspiration is attended with a crowing noise. When the closure is more complete the function of respiration is entirely suspended for a while; there is an effectual obstacle to the admission of air. The child makes vehement struggles, by some termed convulsive, to recover its breath. At varied intervals, from a few seconds up to a minute,

or upon some occasions nearly two minutes, air is at length admitted through the glottis, now partially open; and this rush of air, passing through a very narrow chink, produces the peculiar sound. To these symptoms not unfrequently succeeds a fit of coughing or crying, which terminates the scene; or, if the glottis be not thus partially open, the child, at the end of from two to three minutes at the utmost, will die suffocated. Pallid and exhausted, it falls lifeless upon its nurse's arms; and it is then that the child is generally said to have died in a fit."

We have said already, that paralysis of the branches of the pneumo-gastric supplied to the œsophagus, as well as of those terminal branches furnished to the stomach and intestines, to the liver and other abdominal organs, are pathological results which are known (so far as they are known at all) only as consequences of some disease or pressure upon the trunk of the pneumo-gastric itself (one or both) in some part of its course through the neck or thorax. So now, it is necessary to add that paralysis of the remaining branches of the pneumo-gastric—that is, of the **pulmonary** and the **cardiac branches**—is met with almost solely as consequences of disease or pressure upon the trunk or nucleus of origin of one or other of the pneumo-gastric nerves.

After speaking of the effects of the involvement of the pulmonary and cardiac branches of the vagus, something further will have to be added concerning the causes and most common effects of disease or pressure upon the trunk of either pneumo-gastric nerve.

THE BRONCHI AND LUNGS.

It should be borne in mind that the pneumo-gastric is the chief afferent nerve in connection with the respiratory centre. It seems to convey 'accelerating' and also 'inhibitory' impressions to this centre. The accelerating impressions, as a rule, greatly preponderate, so that experimental division of the pneumo-gastric in an animal causes its respirations to be less frequent but deeper; while stimulation of the central cut end quickens the respiration. Inhibitory impressions are conveyed principally by the fibres of the superior laryngeal nerve, and their experimental stimulation has been found to arrest respiration. But the pneumo-gastrics also supply the motor fibres that innervate the muscles of the bronchi, as well as those of the smaller air tubes throughout the lungs.

In certain typical cases of paralysis of the vagus, observed in the human subject, the respiration has been modified in just the manner noted in animals in whom one of the pneumo-gastric nerves has been cut—that is to say, the respiration has been rendered slower, the inspiratory movements being less frequent but more profound. At other times, however, when there has been good reason for believing in the existence of paralysis of the vagus, there have been paroxysms of dyspnœa, with respirations from 160 to 170 per minute. It seems only possible to explain this state of things, on the supposition that the fibres of the vagus which transmit accelerating impressions to the respiratory centre have been irritated rather than paralysed. It is far less probable that such a condition could have been produced, merely by paralysis of the inhibitory fibres contained in the superior laryngeal nerves. It may be quite true that powerful stimulation of such fibres is capable of arresting respiration, and yet it may not at all follow that the paralysis of these fibres should, of necessity, lead to a marked increase in the rate of respiration.

The pneumo-gastric is believed by many to contain the vaso-motor nerves that are distributed to the vessels of the lungs, as well as certain trophic nerves. Section or severe compression of either of these sets of nerves is said to lead to marked congestion, together with extravasation of serum into the affected organ. Later on, pneumonia and tuberculisation are apt to be met with, and these are regarded by some as trophic lesions, due in some way to the cutting off of the influence of the vagus. Concerning the mode of production of these changes, however, there is much room for doubt, since Brown-Séquard, as well as Franck, have shown that the vaso-motor fibres for the pulmonary vessels are distributed with the sympathetic and not with the vagus. A similar uncertainty exists as to the cause of the so-called trophic changes, although it is undoubtedly true that animals, after section of the vagus, are particularly prone to die from chronic pneumonic or phthisical changes of some kind. It has been suggested (Traube, Steiner), that the pneumonic changes are chiefly determined by passage of food into the bronchi from the pharynx, owing to paralysis of the œsophagus, on the one hand, and on the other, of the sensory and motor fibres of the larynx.

In nervous asthma we have to do with **spasm** of the smaller bronchial tubes, whose muscular fibres are innervated by the pneumogastric. This disease is, in fact, a neurosis connected with a partially disordered activity on the part of the vagus and its central nucleus. Although individual attacks may be excited by irritant particles, gases, or the air of this or that place, still, the diseased condition itself is a neurosis dependent upon certain unnatural molecular conditions or states of the nerve centres, the tendency to the establishment of such conditions being often inherited.

At times, the pressure of mediastinal tumours or aneurysms upon the branches of the pneumo-gastric in the pulmonary plexus, is found to give rise to paroxysmal attacks of cough and dyspnœa of an asthmatic character, which attacks are often accompanied by the abundant secretion of a thin watery sputum.

THE HEART.

In cases in which the vagus has been cut in one of the lower animals, the effect upon the heart-beats is just the reverse of that which is produced upon the respiratory movements—whilst the respiratory movements are made distinctly slower, those of the heart are even more distinctly increased in frequency.

The same thing has been observed clinically, in patients suffering from paralysis of the vagus. Hayem has recorded a case in which the heart beat from 120 to 160 a minute; and in another case, observed by Tuczek, the pulse varied from 190 to 208 per minute. Again, in a case of phthisis, recorded by Meixner, in which the pulse was at first occasionally, and afterwards constantly, noted to range from 130 to 140, the left vagus was subsequently found to be enclosed in a mass of enlarged glands near the upper margin of the thorax.

Any irritation of the trunk of the vagus which affects its cardio-inhibitory fibres, leads, when moderate in amount, to a slowing of the heart's action; or, when it is more marked, to a complete arrest of cardiac action.

The vagus also contains fibres that convey afferent impressions of an inhibitory type to the principal vaso-motor centre. The advent of such impressions in excess lowers the activity of the vaso-motor centre, and, as a consequence, some amount of dilatation of the small arteries occurs throughout the body.

In some cases the occurrence of angina pectoris, which has long been known to be an affection intimately related to modes of disordered activity in the vagus and cardiac branches of the sympathetic, has been found to be distinctly associated with the existence of tumours (Heine, Blandin) involving either the cardiac plexus, or some part of the trunk of the vagus itself.

CAUSES OF DISEASE IN THE TRUNK OF THE PNEUMO-GASTRIC NERVE.

We have now dealt in detail with the signs and symptoms, referable to different organs or parts, liable to be caused at different times by disease or injury to the pneumo-gastric nerves. We have referred to the etiological problems, or those connected with pathological diagnosis as yet, only in relation with certain branches, such as the pharyngeal and the laryngeal, which are most apt to be implicated separately (see pp. 469, 474). We must now, very briefly, refer to the various causes that lead to involvement of the trunk of the pneumo-gastric itself, below the point at which the superior laryngeal nerve is given off (that is, below the level of the second or third cervical vertebra). This limitation is made in order not to go over ground which has been already considered.

Guttmann has well classified the organic causes interfering with the functions of the pneumo-gastric nerves into three categories :—compressions, traumatisms, diseases of the nerve.

(1).—**Compression** seems to be the most frequent cause; this being brought about by tumours growing in the neck or in the mediastinum. These are represented by enlarged lymphatic glands, cervical or bronchial; and also by aneurysms of the carotids, subclavians, or of the aorta itself. (Aneurysms of the transverse or descending part of the arch of the aorta are specially apt to involve the left recurrent laryngeal nerve.)

(2).—Under the head of **traumatisms**, we may mention injuries to the trunk of the vagus caused by bullet-wounds or by stabs; also accidental injuries caused by surgeons, during the removal of tumours or enlarged thyroid bodies (in the latter case the recurrent laryngeal branches are also specially apt to be involved). Again the trunk of the vagus has been tied during ligation of the common carotid artery.

(3).—The cases coming under the head of **diseases of the vagus** are extremely rare. Still, the nerve is occasionally the seat of neuromata, and very rarely, also, it may be the seat of some chronic inflammatory process.

The special and general causes tending to affect the roots of the nerve, or its nuclear centre in the medulla, have already been referred to. Such causes are, in part, common with those that involve the roots and centre of the accessory portion of the spinal accessory nerve.

I.—THE SPINAL ACCESSORY NERVE.

The upper part of the spinal accessory nerve emerges from the surface of the medulla in a line with, and just below, the pneumo-gastric nerve (Fig. 107); while the nucleus of origin of this part of the nerve, consists of a column of nerve cells also situated in a line with and below that of the pneumo-gastric—both of them lying just outside the nucleus of the hypoglossal nerve (Fig. 108). These upper fibres of the spinal accessory join the pneumo-gastric (Fig. 109), and go to their destination mixed up with its fibres; as already indicated, they supply the muscles of the larynx, part of the pharynx, and in all probability, also, the levator palati. The effects and causes of paralysis of this accessory part of the spinal accessory nerve have already been dealt with, in the last section (pp. 469, and 474).

The lower fibres of the spinal accessory nerve emerge from the lateral column of the spinal cord as low down as the sixth or seventh cervical nerves, taking their origin from the anterior cornua. The several fibres of this spinal portion of the nerve pass upwards through the foramen magnum, joining the accessory portion (Fig. 109) for a short distance before uniting with the pneumo-gastric. It then passes down the neck, where its fibres innervate the sterno-mastoid and also the upper part of the trapezius muscle.

Paralysis of the spinal accessory nerve is rarely total; it can, indeed, only be so (that is without coincident paralysis of the pneumo-gastric) by the occurrence of lesions in two situations—either (*a*) in the very limited part of its course in which the accessory and the spinal divisions of the nerve are in contact with one another, or (*b*) by the simultaneous implication of its nuclei of origin in the medulla and in the spinal cord. Implication of the total trunk of the nerve is excessively rare; on the other hand, the implication of both the medullary and the spinal nucleus simultaneously is by no means uncommon, though brought about by one pathological condition only, viz., that degenerative process which affects the motor ganglion cells in 'progressive muscular atrophy,' and in its bulbar representative 'labio-glosso-laryngeal paralysis.'

Paralysis of the accessory portion of the nerve is met with principally in cases of disease involving the trunk of the pneumo-gastric. It may, therefore, of course, be involved, quite independently of the spinal portion. At other times, paralysis of the accessory portion of the nerve is induced by localised lesions involving its nucleus in the medulla oblongata, though in such a case there would almost certainly be other coexisting evidence of centric disease.

Paralysis of the spinal portion of the nerve, alone, occurs not unfrequently; and then, either the whole, or only a part of its fibres may be involved. It passes through the jugular foramen on the outer side of the pneumo-gastric and within the same sheath. It then passes backwards, behind this nerve and the jugular vein, to reach the upper portion of the sterno-mastoid. After furnishing many branches to this muscle (which is supplied to some extent also by branches of the third cervical nerve), it pierces it, crosses the posterior triangle of the neck, and is distributed to the under surface of the upper part of the trapezius (other parts of this muscle being innervated by branches from three or four of the cervical nerves).

Paralyses of this spinal part of the nerve may be caused in various ways. Among such causes may be enumerated disease of the occipital bone, fractures of the cervical vertebræ, tumours, swellings of lymphatic glands, abscesses, neuritis of rheumatic or syphilitic origin, or traumatic injuries of different kinds (stabs, gunshot wounds, etc.).

Where we have to do with severe compressing or destructive lesions, the sterno-mastoid and the trapezius may be paralysed, separately or conjointly according to the situation of the lesion in question.

Unilateral paralysis of the sterno-mastoid muscle, in consequence of the unopposed action of the muscle on the opposite side, causes the head to be turned to the affected side with the chin somewhat elevated. It cannot be easily rotated in the opposite direction by an effort of the will, though passive movement in this direction is easily brought about. When the chin is supported by the hand, and the patient is directed to move it forcibly downwards or laterally, the sterno-mastoid on the sound side alone projects, or, when the paralysis is partial, projects much more than on the opposite side. When a unilateral paralysis has lasted for a long time, secondary contracture of the healthy muscle occurs, and gives rise to an exaggerated and persistent obliquity in the position of the head.

With bilateral paralysis of the sterno-mastoid muscles, the head is maintained in a straight position with the chin slightly elevated, but it cannot be easily rotated to either side. There is not only an absence of prominence on either side, when attempts are made to call the sterno-mastoids into activity, there may even be some amount of actual hollowing of the neck in the situations naturally occupied by these muscles, when anything like marked atrophy has occurred. Atrophy is, moreover, prone to be present in these cases of bilateral paralysis of the sterno-mastoids, because such a condition is most likely to be due to centric disease of a degenerative type affecting the ganglion cells of the spinal nuclei.

Unilateral paralysis of the trapezius is principally revealed by the unnatural position assumed by the scapula. It is drawn somewhat downwards and forwards, and its inner border becomes oblique, so that the inferior angle is slightly approximated to the spinal column. The acromion falls downwards and somewhat forwards—partly owing to the weight of the arm, and partly because of the now unbalanced action of the rhomboideus and levator anguli scapulæ. As a consequence, the clavicle projects, the supra-clavicular fossa appears deeper than natural, and the postero-superior angle of the scapula can be felt with unnatural distinctness.

Paralysis of the upper third of the trapezius principally interferes with elevation of the shoulder (though after a time the levator anguli scapulæ takes on extra functions in this direction, and may become distinctly hypertrophied). It also interferes with elevation of the arm above the horizontal position. This latter action is disturbed, because in the absence of the traction exercised upon the acromion by the upper third of the trapezius, the elevating action of the deltoid and of the serratus magnus are much interfered with.

With bilateral paralysis of the trapezii the characters already mentioned are present on both sides. The back assumes a peculiar appearance, owing to the falling forwards and outwards of both shoulder blades ; the head is prone to fall forwards on the chest, and there is difficulty in maintaining it in a straight, upright position.

Where the whole trunk of the spinal branch of the nerve is affected, we have simultaneous paralysis of the sterno-mastoid and of the trapezius, and, consequently, a combination of the signs above described for paralysis of these muscles separately. Should the entire spinal accessory nerve be damaged just outside the jugular foramen, in the situation where the accessory and the spinal portions of the nerve come together, then we should have, in addition, signs of paralysis of the accessory branch, viz., some hoarseness, nasal quality of voice, and difficulty of swallowing (see p. 469). This, however, as we have already indicated, is an extremely rare combination.

The recognition of the precise kind of paralysis affecting the neck and shoulder muscles often requires much care. Where the defect is unilateral, most careful comparisons must be made of the movements of the head and of the shoulder blade on the two sides. In order to determine whether we have to do with paralysis of this or that muscle on one side, or with tonic spasms or contractures of the antagonist muscles, we form our opinion from the freedom or the reverse of passive movements in this or that direction; we may also often be guided by the results of faradic excitation of the muscles in question.

Spasms, either tonic or clonic, may occur in the muscles supplied by the spinal branch of the spinal accessory nerve.

Tonic spasms are the most frequent, and they produce the rather common condition known as torticollis or wry-neck.

This form of spasm, is most frequently limited to the sterno-mastoid on one side, much less frequently to one trapezius, while occasionally both muscles may be simultaneously affected.

In tonic spasm of one sterno-mastoid the chin is turned to the opposite side and raised, the ear on the affected side being approximated to the clavicle; the rigid muscle forms a hard projection; and, owing to the spasm, the faulty position is difficult to rectify either by passive movements or by the patient's voluntary efforts. Slight pains may be experienced for a time, but these soon subside. If this condition persists for a long time, two consequences may at last show themselves; first, more or less atrophy of the antagonist muscles, and, secondly, especially in young persons, a curvature of the spinal column with its convexity towards the sound side.

Tonic spasm of both sterno-mastoids is excessively rare. In such a case the head is drawn forwards, and depressed upon the chest.

When tonic spasm of the upper third of the trapezius occurs, the head is persistently drawn towards the affected side; and the occiput is approximated to the shoulder, which is somewhat raised, whilst the scapula is drawn slightly inwards. The anterior border of the affected trapezius forms a hard swelling. The resistance to passive movements, further distinguishes this form of displacement from that caused by paralysis of the opposite sterno-mastoid.

In other forms of torticollis or wry-neck, we have to do with **clonic spasms** of the same muscles, affecting them either separately or in combination.

Where the clonic spasms are limited to one sterno-mastoid, the head is moved convulsively backwards and to one side, in the direction above described under the head of tonic spasm of this muscle. We have now, however, a succession of momentary spasms, rather than one enduring contraction.

The same kind of thing occurs in the rarer cases in which there are bilateral clonic spasms of the sterno-mastoids. The head, instead of being persistently drawn forwards and downwards, is now affected with forward and backward nodding movements. This condition is met with more frequently in children than in adults.

When the upper third of one trapezius is the seat of clonic spasms, we have a succession of movements occurring in the direction indicated under the head of tonic spasm of this part of the trapezius. At times, the head may be drawn backwards with such force that the occiput and shoulder almost touch.

Where both the sterno-mastoid and the trapezius of one side are affected, the contractions of the two muscles may alternate with one another; or the spasms of both muscles may occur coincidently, and then the resulting movements will naturally vary within certain limits, according as differences occur in the respective strength of the spasms involving the two muscles.

In these various cases, the clonic spasms at first show themselves in distinct paroxysms, with longer or shorter intervals of comparative quietude. As time goes on, or, in more severe cases, from the first, the spasms become more continuous—undergoing little cessation, it may be, except during sleep. When the spasms occur only in paroxysms, they are always exaggerated by mental excitement, or by the patient's consciousness that he is being observed—as under any other conditions productive of 'nervousness.' In the more severe cases, too, other cervical muscles are apt to share in the spasms; especially when such spasms are dependent upon altered functional states of the ganglion cells constituting the nuclei of the spinal portion of the accessory nerve. This is scarcely to be wondered at, when we consider that the cervical nerves, innervating the other cervical muscles, arise from regions of the anterior cornua closely contiguous to those giving origin to the roots of the spinal portion of the spinal accessory nerve.

Sometimes spasms occurring in the muscles supplied by the accessorius, are at first of the clonic order; and, after a time, these may give place to tonic spasms with permanent contracture in the same muscles.

In regard to **diagnosis**, it is most important not to confound cases of disease of some of the cervical vertebræ associated with unnatural and fixed positions of the head and neck, with cases of tonic spasm of muscles supplied by the accessorius.

Again, for the recognition of the true nature of the affections brought before us, it is most important to bear in mind the fact that tonic spasms of the muscles to which we have been referring, may be confounded with paralysis of their antagonists, unless due regard be had to the fact that the latter conditions do not, like the former, interfere with the freedom of executing passive movements.

The pathological diagnosis of the spasms in question, is often a matter of extreme difficulty, on account of the very varied nature of the causes or antecedent conditions preceding the onset of such spasms.

We must content ourselves with a brief enumeration of the principal of these causes.

The incidence of cold, leading to rheumatic inflammations of the muscles, or muscles and peripheral portions of the spinal accessory nerves. Traumatisms, or diseases of any kind (such as tumours, abscesses, etc.), which cause irritation of the trunk of the nerve in any part of its course. Disease of the cervical vertebræ (caries, periostitis, or tumours) may irritate the cervical region of the cord itself, or the roots of the spinal accessory outside the cord. Cervical meningitis or tumours in the cervical region of the cord, may similarly irritate these nerve roots.

In addition to these more direct physical causes of irritation of the spinal accessory nerve, there is reason to believe that in other cases spasms of the muscles supplied by the accessorius may be of reflex origin, due to irritations of the dental, the intestinal, or the uterine nerves.

In still other cases, these spasms may occur after some marked emotional disturbance, during the puerperal period, or in the period of convalescence from typhoid or some other severe specific fever.

J.—THE HYPOGLOSSAL NERVE.

The hypoglossal is the motor nerve which supplies the tongue and most of the muscles attached to the hyoid bone.

Paralysis of the tongue (glossoplegia) may be either unilateral or bilateral, and may be either partial or complete.

Such paralysis may be due either (1) to disease implicating its intracerebral fibres above the medulla; (2) to disease involving its nucleus of origin or root fibres; or (3) to pathological conditions affecting its fibres in some part of their course between the surface of the medulla and the tongue.

(1).—In this class of cases, the paralysis is almost invariably unilateral and partial, and associated with more or less of a hemiplegic condition on the same side of the body. The chief sign of this kind of paralysis is that when the tongue is protruded it deviates towards the paralysed side, owing to the genio-glossus being paralysed or weakened on this side, and unable, therefore, to prevent the deviation caused by the stronger action of its fellow on the opposite side. Where the paralysing lesion is situated low down, that is, in the pons Varolii, the paralysis of the tongue is more apt to be severe and bilateral. It may, in this case, be difficult for the patient to protrude its tip beyond the mouth, and no notable deviation to one side or the other may be recognizable; at the same time, deglutition may be difficult, and the patient's power of distinct articulation extremely impaired. In this latter class of cases, the paralysis is apt to last long; but where it is unilateral and less marked (produced by lesions higher up in either hemisphere), it is usually a much more transitory sign.

(2).—Where disease exists in the medulla oblongata, so situated as to involve the nuclei of the hypoglossal nerves (Fig. 108, xii), the paralysis is commonly bilateral, complete, and after a time associated with more or less marked wasting. Atrophy may, however, at times exist and yet be obscured by a coincident or secondary fatty overgrowth. In these cases the tongue lies motionless behind the teeth—being either broad and flabby; or having its substance wasted, wrinkled, and showing more or less continuous flickering contractions.

Where such paralysis is caused by a degenerative process in the medulla, leading to what is known as 'progressive bulbar paralysis,' the lips, palate, pharynx and larynx, are also rendered more or less powerless. But, even without these aggravating adjuncts, the mere paralysis of the tongue causes great difficulty in mastication, deglutition, and articulation. The latter defect shows itself, at first, in the pronunciation of *s*, *sch*, *l*, *e*, and *i*; and, later on, in that of such consonants as *k*, *g*, and *r*. Finally, the patient's utterance becomes almost unintelligible.

The hypoglossal nuclei of one or both sides may, also, be affected by tumours, patches of sclerosis, or small focal lesions caused by hæmorrhage or softening, involving the posterior part of the medulla near the lower end of the fourth ventricle.

The hypoglossal nerves may likewise be affected on one or both sides, but, more likely, on one side only, in some part of their course from their nuclei of origin to the surface of the medulla (Fig. 113, H). They are here apt to be involved more especially by tumours, patches of sclerosis, or foci of softening.

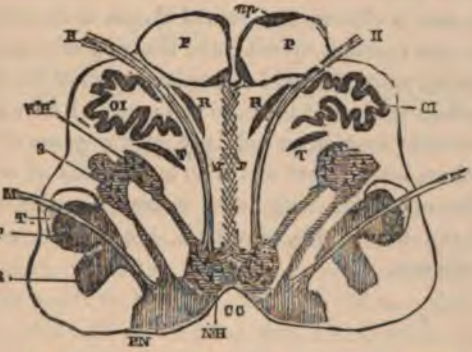

FIG. 113.—DIAGRAM OF A SECTION THROUGH THE MEDIAN PORTION OF THE BULB [after M. Duval.]

PP, Anterior pyramids. CC, Floor of the 4th ventricle. H, Root fibres of the hypoglossal nerves. NH, Nucleus of the hypoglossal; N'H', accessory nucleus of the same. S, Accessory (motor) nucleus of the mixed nerves; PN, principal (sensory) nucleus of the mixed nerves (glosso-pharyngeal, vagus, and spinal accessory). M, Root fibres of the vagus.

NR, Nucleus of the restiform bodies. CP, Head of the posterior cornu. T, Ascending root of the trigeminus. OI, Olivary body. R, Internal, and T, external grey nucleus near the olivary body. *xx*, Raphé.

(3).—The more strictly peripheral portion of the hypoglossal nerve is also apt to be affected by lesions or injuries of various kinds, in its course from the medulla to the tongue. The paralysis in such cases is almost always unilateral. It may exist alone or without such accompaniments as have been specified in either of the categories above referred to. It is apt to become complete for the one-half of the tongue, and to be associated with atrophy and superficial flickerings.

The pathological conditions most apt to affect the hypoglossal nerves themselves are, tumours involving the surface of the medulla; chronic meningitis; syphilitic thickenings or growths; fibroid adhesions between it and the aneurysmally dilated walls of a vertebral artery (as I have myself seen); or caries of the first cervical vertebra. Outside the skull, the nerve may become involved in tumours of different kinds, or may be affected by traumatic causes.

Spasms of the muscles supplied by the hypoglossal nerves are rarely met with as independent affections, though, either in the tonic or the clonic form, it is possible that they may be produced by lesions of an irritative type involving its nuclei or fibres in the medulla.

On the other hand, as components of other morbid states, spasms of the muscles supplied by the hypoglossal nerve, of the tonic or clonic variety, are of very common occurrence. Some years ago I saw a remarkable case of chronic and partial chorea in an elderly woman, in which there was constant opening and shutting of the mouth together with protrusion, retraction, and other movements of the tongue, as well as certain clonic spasms of the head and neck. I have recently seen a somewhat similar case in an old lady, eighty years of age. Again, spasms of these muscles are met with in all forms of convulsions; or they may be associated with facial spasms or with trismus. Mere tremors of the tongue are, of course, apt to occur under the most various conditions.

PART IV.

PARALYSES OF SPINAL ORIGIN.

PRELIMINARY DATA.

The Spinal Cord extends through the spinal canal, from the level of the upper border of the body of the first cervical vertebra, as far as the upper part of the body of the second cervical vertebra. It is continuous with the bulb or medulla oblongata above; while below, it tapers conically, and ends in a slender filament, known as the 'central ligament' of the spinal cord.

Within the spinal canal, the cord is enveloped in a loose sheath formed by the dura mater; the latter being separated from the unyielding walls of the canal by venous plexuses, and much loose areolar

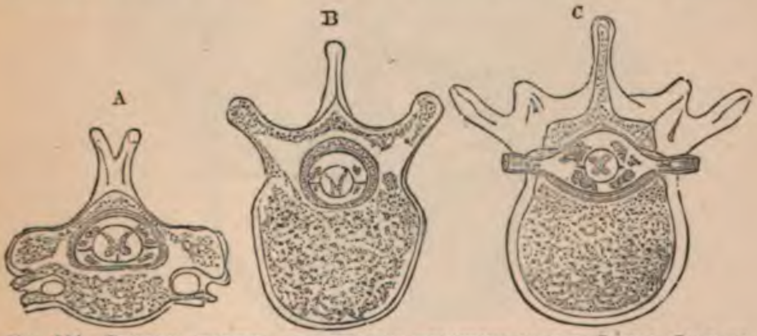

FIG. 114. SECTION SHOWING THE GENERAL RELATIONS OF THE SPINAL CORD TO THE INCLOSING THECA, AND OF THIS TO THE VERTEBRAL CANAL [Key and Retzius].

A, Section through the fifth cervical vertebra; B, through the tenth dorsal vertebra; C, through the first lumbar vertebra.

In each section, the cord, covered by pia mater, is seen in the centre, with the ligamentum denticulatum attached to it on either side; the nerve roots on each side form small groups which, since they pass obliquely downwards to their foramina of exit, are cut across; the dura matral sheath is separated by a considerable space from the cord, and also by a quantity of loose areolar and fatty tissue from the wall of the vertebral canal. This latter tissue is in smaller amount in C. The arachnoid is not represented in these sections.

tissue containing fat. The cavity of the theca is occupied by cerebro-spinal fluid. This cavity is divided by the arachnoid into two spaces, the 'subdural' and the 'subarachnoid spaces'; the latter being much the larger of the two, and containing the fluid above referred to, which immediately surrounds the spinal cord with its closely investing pia mater. The cord is kept in position by a ligament on each side (ligamentum denticulatum) which fixes it at frequent intervals to its sheath, and also by the roots of the spinal nerves, passing from the surface of the cord towards the intervertebral foramina. The uppermost nerve roots cross this space nearly horizontally, but the rest pass across with a more and more oblique downward inclination, until, when we come to the lumbar and sacral nerves which go to form the so-called cauda equina, their direction is almost vertical.

The cervical swelling of the cord commences at the level of the third cervical vertebra; it increases in size up to the fifth; preserves the same calibre to the sixth cervical; and then gradually diminishes as far as the second dorsal vertebra. From the second to the tenth dorsal vertebra the cord preserves the same diminished size. Opposite the tenth dorsal vertebra it begins to swell again (though to a much less extent) into the lumbar enlargement, which, as already stated, terminates opposite the first or second lumbar vertebra in the central ligament, by which the lower extremity of the cord is kept in position.

According to Sappey the mean diameter of the spinal cord is to that of the vertebral canal as 3 : 5.

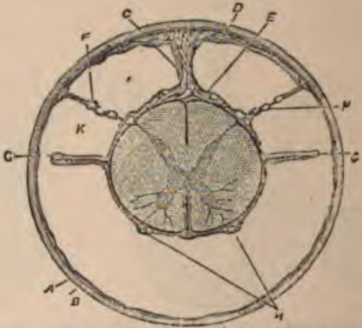

FIG. 115. ENLARGED TRANSVERSE SECTION OF THE SPINAL CORD AND ITS MEMBRANES IN THE UPPER DORSAL REGION [after Key and Retzius].

Close to the inner surface of the dura mater (A) lies the arachnoid (B), which is thrown into longitudinal folds at intervals. The ligamenta denticulata (G,G) divide the sub-arachnoid space into an anterior and a posterior division, and in the latter may be seen the septum posticum (C), and also the posterior nerve roots (F,F). The septum posticum becomes partly attached to the arachnoid externally, and partly spreads laterally over the inner surface of that membrane (D); it also spreads internally over the pia mater as the epi-pial subarachnoidal tissue (E). The anterior subarachnoid space may be seen to be free from intersecting trabeculæ. H, are the anterior nerve roots seen in transverse section.

The spinal **pia mater** is a much denser and firmer membrane than that which surrounds the encephalon; it is also less vascular and more closely adherent to the subjacent nervous matter by means of prolongations into its substance. It has even been described by some writers under a different name, as the neurilemma of the cord.

The **spinal arachnoid** is a thin membrane quite distinct from the spinal pia, and separated from it by a considerable distance. The only approximation to such an arrangement to be found within the cranium is that which exists in the great subarachnoid space at the base of the brain. Fine trabeculæ of connective tissue stretch through the cerebro-spinal fluid in this great spinal subarachnoid space. They are met with principally in the posterior division of the space; some ensheath bloodvessels, and others surround the posterior roots of the spinal nerves.

The spinal **dura mater** is not closely adherent to the walls of the vertebral canal. Posteriorly it is surrounded by a plexus of veins, and is also separated from the vertebral arches and the ligamenta subflava by a quantity of areolar

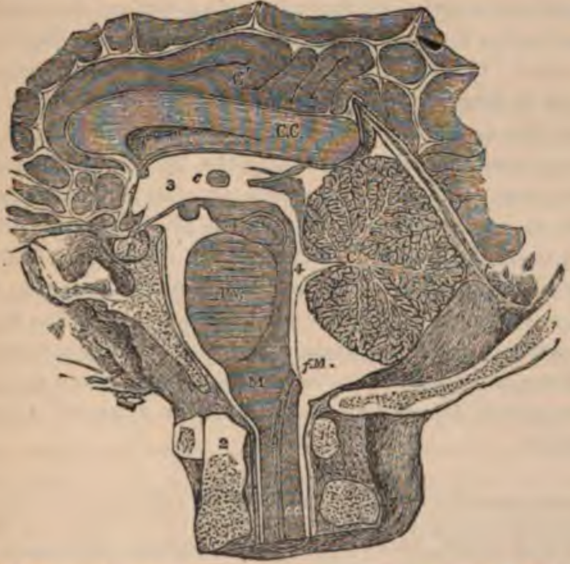

FIG. 116. SECTION OF THE POSTERIOR AND LOWER PARTS OF THE BRAIN WITHIN THE SKULL, TO SHOW THE SUBARACHNOID SPACE (CEREBRAL AND SPINAL), AND ITS RELATIONS TO THE VENTRICLES [after Key and Retzius].

The section was made in the frozen state, the cavities having been previously filled with injection.

1,1′, Atlas vertebra. 2, Odontoid process of the axis. 3, Third ventricle. 4, Fourth ventricle. C,C, Corpus callosum. C′, Gyrus fornicatus. C, Cerebellum. *t*, Tentorium. *p*, Pituitary body. *c,c*, Central canal of the cord.

f M, in the cerebello-medullary part of the subarachnoid space, is close to the foramen of Majendie, by which that space communicates with the fourth ventricle. It will be seen also that the encephalic subarachnoid space is directly continuous with the spinal subarachnoid space.

tissue containing a reddish coloured fat. Anteriorly it is adherent by fibrous tissue to the posterior common ligament. Such adhesions are scanty in the dorsal region; are numerous and long in the lumbar region; but are still more numerous and much shorter in the cervical region. These adhesions become most intimate at the level of the second cervical vertebra. Above, the spinal dura mater gradually passes into the cerebral dura mater. The spinal dura mater is a distinctly elastic membrane, a fact which is of some importance in connection with its functions as a reservoir of cerebro-spinal fluid.

It is important to recollect that the subarachnoid space around the cord is directly continuous with the subarachnoid space around the encephalon; and that the latter is in communication, through the foramen of Majendie, with the fourth ventricle and, of course, through it, with the third and lateral ventricles. The perivascular canals in the encephalon and spinal cord, may also be regarded as so many prolongations of their common subarachnoid space.

VASCULAR SUPPLY OF THE SPINAL CORD.

The principal arteries supplying the spinal cord are derived from the vertebrals in the manner which has been already described (p. 327). They consist (1) of two anterior spinal branches, which soon unite so as to form a single median branch running along the anterior aspect of the cord; and (2) of two posterior spinal arteries which run down the cord just behind the line of attachment of the posterior roots. In their course these principal arteries are joined by a number of small twigs derived from the inferior thyroid arteries in the neck, from the intercostal arteries in the back, and, lower down, from the lumbar arteries. The three main vessels anastomose with one another, and they give origin to a number of lateral branches which also freely anastomose with one another in the pia mater surrounding the cord. From this superficial network of branches, vessels are given off for the supply of the substance of the cord in a manner presently to be described.

Some very suggestive remarks were made a few years ago by Dr. Moxon concerning the blood supply of the cord, and with the view of explaining the special frequency with which softening implicates the lower portion of this organ. He says ('Brit. Med. Jrnl.,' April, 1881):—"The blood supply to the spinal cord is carried out by slender vessels which come from the vertebral arteries within the cranium. There are three of these arteries—one on the front and two on the back of the cord; they are very slender, and yet have to run along its whole length. No arteries so small as these run so great a length elsewhere in the body, and pressure falls rapidly in minute arteries as the length of pipe increases, so that it becomes necessary to reinforce these slender vessels wherever possible; and advantage is taken of the nerve-roots to send up little reinforcing arteries along these. In the part of the cord corresponding to the neck, upper extremities, and trunk, where the nerve-roots are short, the reinforcing arteries are also short, and they reach, and join, and furnish blood to the spinal arteries—so that in this part of the cord every segment of it is supplied with blood from two directions, the anterior spinal artery bringing blood from above and the reinforcing artery from below. But when you approach the tip of the cord the supply from below becomes exceedingly precarious, and even apt to fail entirely, because upon the long strands of the cauda equina the small arteries are too narrow and too long to reinforce the cord with any certainty. But, at the same time, the supply from above has to be furnished with greater difficulty than in the upper regions of the cord, because the original anterior spinal artery is very far away and the reinforcing arteries even in the lumbar region have to run considerably longer courses than they had in the cervical region. . . . . Hence we see that the tip of the spinal cord, corresponding to the lower limbs and sphincters, is much more weakly organised as to its circulation than are the upper parts of the cord."

The vessels that supply the substance of the spinal cord are, according to the description which we borrow from the ninth edition of Quain's 'Anatomy' (Vol. i, p. 380), distributed in the following manner:—

"The small entering branches may be described as forming two systems, a centrifugal and a centripetal. The first is composed of a series of arterioles, 250 to 300 in number, which pass from the anterior spinal artery into the anterior median fissure, penetrating to the anterior commissure. Here each one divides into a right and left branch, which again divide into smaller arteries and capillaries for the central part of the corresponding crescent of grey matter; but an ascending and a descending ramuscle is also given off on either side, for anastomosis with the corresponding vessels above and below."

"The second or centripetal set have a converging or radial arrangement, passing in from the periphery. Some of these simply form capillary loops, which supply the superficial layers of the cord. Others are distributed to the white matter, where they form comparatively large meshed longitudinal plexuses. But the most considerable of the centripetal arteries penetrate to the grey matter, and pour their blood into the close capillary network which pervades it, supplying the parts not served by the centrifugal vessels. Special mention may be made of a series of small median arterial branches which enter the posterior fissure, penetrating in it to the posterior commissure, and giving off branches which supply the adjacent parts of the posterior white columns and Clarke's column, where this is found; and of the vessels which enter the grey matter with the bundles of the anterior and posterior nerve roots, and are distributed to the corresponding cornua.

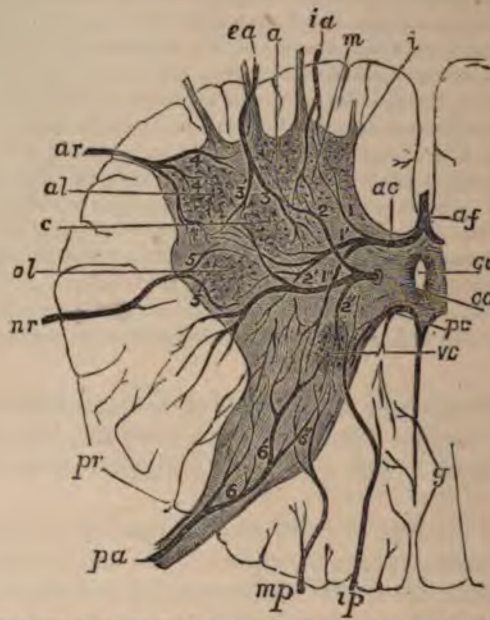

FIG. 117. DIAGRAM SHOWING THE DISTRIBUTION OF THE BLOOD-VESSELS IN THE SPINAL CORD [after Ross].

Anterior median artery.
  af, Arteries of the anterior median fissure.
  ac, Artery of the anterior commissure.
    1, Anterior branch.
    1′, Median branch.
    1″, Posterior branch.
  ca, Central artery.
    2, Anterior branch.
    2′, Median branch.
    2″, Posterior branch.
na, posterior root arteries.
6, 6′, 6″, Arteries of posterior horns.
ia, Internal anterior root artery.
ea, External anterior root artery.
  3, 3′, Internal and external branch.
ar, Antero-lateral branch.
  4, Anterior branch.
  4′, Median branch.
  4″, Posterior branch.
mr, Median lateral artery.
  5, 5′, Anterior and posterior branches.
pr, Posterior lateral arteries.
ip, Internal posterior artery.
mp, External posterior artery.
g, Arteries of the column of Goll.
pc, Artery of the posterior cornu.
vc, Vesicular column of Clarke.
i, Internal group of cells.
a, Anterior group.
al, Antero-lateral group.
pl, Postero-lateral group.
c, Central group.
m, Median area.

"The veins run alongside the arteries. Two longitudinal venous vessels, one on either side of the central canal, accompanying the corresponding anastomotic arteries, are conspicuous in most transverse sections of the cord."

The common situations of, and the names proposed for, the different arterioles of the cord may be seen by reference to Fig. 117.

OTHER ANATOMICAL, TOGETHER WITH SOME PHYSIOLOGICAL, DATA CONCERNING THE SPINAL CORD.

It seems essential that certain anatomical and physiological facts concerning the spinal cord—beyond the question of its general relations and vascular supply already referred to—should be mentioned as a preliminary to the consideration of its various diseases.

Reference must be made especially (*a*) to the grouping of ganglion cells in its grey matter; (*b*) to the mode of origin or distribution of the fibres of the anterior and posterior roots in the substance of the cord; (*c*) to the fundamental tracts into which the different columns of the cord may be divided; and lastly (*d*) as to the nature of the functions subserved by these various columns and parts of the spinal cord.

(a).—Grouping of Ganglion Cells in the Grey Matter of the Cord.

The ganglion cells in the spinal cord are collected together into groups, and the distribution of these is subject to certain variations at different levels or segments of the cord.

The useful diagram of Ross (Fig. 117) will suffice to give a good notion as to their distribution in certain important parts of the spinal cord. Though it is not an accurate representation of any one section, it more closely resembles, as he says, what would be met with in the upper part of the lumbar and the lower part of the cervical enlargement, than in a section of any other region.

In the posterior cornua the cells (comparatively small and spindle-shaped) are, for the most part, diffusely scattered, rather than aggregated, into distinct groups.

At the internal part of the base of the posterior cornu, on each side, there is however a distinctly defined group of cells (*vc*), commonly known as Clarke's column. This is only present in certain parts of the cord. It begins to be found at the upper end of the lumbar enlargement, is continued throughout the dorsal region, and ceases again at the lower part of the cervical enlargement. It seems to be represented also in the medulla.

Concerning this column there is the following statement in Quain's 'Anatomy' (9th Ed., Vol. II., p. 273):—"A second very well-marked group or column of cells, which occupies in transverse section an area at the inner or mesial angle of the base of the posterior grey cornu, and appears cut off from the rest of the grey matter by a curved bundle of fibres derived from the posterior root, extends along the middle region of the cord from the third lumbar to the seventh cervical nerve. This was termed by Lockhart Clarke the posterior vesicular column; it is often known as Clarke's column. It is largest in the lower part of the dorsal region. From the fact that it is almost entirely confined to that region of the cord it was termed by Stilling the dorsal nucleus. But although ceasing above and below at the points mentioned, it is not altogether unrepresented in other parts, for groups of cells are found in a similar situation opposite the origin of the second and third sacral nerves (sacral nucleus of Stilling) and opposite the origin of the third and fourth cervical nerves (cervical nucleus); and elsewhere there are scattered cells in the same part of the section of the cord. The cells of this column are of moderate size, and their axis-cylinder processes tend for the most part towards the lateral column. It is noteworthy that Clarke's column is best developed in the region of the cord where the column of the anterior cornu is least so."

At the inner side of the anterior cornu we meet with the internal group (*i*) of large ganglion cells; just outside these is the anterior group (*a*); whilst at the lateral aspect of the anterior cornu we meet with two other large groups—the antero-lateral (*al*), and postero-lateral (*pl*) groups of cells. Inside these and near the centre of the cornu is the central group (*c*). The relations of the blood-vessels to these several groups of cells may be seen by a careful study of the same figure.

To another group Ross has called special attention. It lies between the internal group, on the one hand, and the antero-lateral and central groups on the other. He says, "The cells of this median area (*m*) are much smaller than those of the other groups, and the area itself is exceedingly vascular, being supplied by the two anterior root arteries, the anterior branch of the first division of the artery of the anterior median fissure, and the anterior branch of the central artery."

The nomenclature here followed is that of Erb slightly modified. Ross says ('Brain,' April, 1880):—"The group which Erb calls anterior, I propose to call internal (*i*), because a very constant group of caudate cells is always observed near the anterior roots, which from its position is best distinguished as the anterior group (*a*). Also the group which Erb calls the median, I propose to call the central (*c*), because I wish to distinguish a very important area (*m*), which has hitherto not been described, and which from its position and connections is best denominated the median area."

Concerning this median area Ross adds the following interesting remarks and speculations:—"The cells of the median area are caudate, but they are much smaller than those of the other groups. This area is only present in the cervical and lumbar enlargements, the portions of the cord which regulate the movements of the limbs; its relatively larger size differentiates the anterior grey horn of the cervical enlargement of the adult human cord from the lumbar enlargement; in the human embryo at the fifth month and in the cord of the ox and dog, this area is of the same relative size in both the cervical and lumbar enlargements, and the anterior horns are under such circumstances almost indistinguishable; hence it may be presumed that in the median area are mainly organised the complicated movements which distinguish the hand of man from the anterior extremity of the lower animal, so far as these movements are represented in the spinal cord. Other interesting facts tending to the same conclusion are that the cells of the median area in the human cord only assume processes after birth; while those of the other groups possess distinct processes at the fifth month of embryonic life.'

"The median area is entirely absent in the upper cervical and dorsal portions of the cord, and consequently the internal, anterior, antero-lateral, and central groups approximate, so that they are not always readily distinguishable. In these regions, however, an area is interposed in the human cord between the antero-lateral and postero-lateral groups, which possess some of the characteristics of the median area in the cervical region. The cells of this area are relatively small, they are comparatively late in development, and do not possess distinct processes at birth; and the area is entirely unrepresented in the cord of the ox and dog; hence it may be inferred that this area represents the additional organisation rendered necessary by the maintenance of the erect posture in man."

" . . . . It is also worthy of remark that the development of the cells proceeds from the centres of the groups to their margins, and that in progressive degenerative diseases, the degeneration proceeds from the margins of the groups to their centres. To this law the median area in the lumbar and more especially in the cervical enlargement and that which lies between the antero-lateral and postero-lateral groups in the dorsal and upper cervical regions, are apparent exceptions. These areas although containing cells, can hardly be called groups; they are probably areas where the marginal cells of the real groups meet. But whatever be their nature they are the most vulnerable portion of the cord, and as they are the last to be developed, so they manifest the greatest liability to disease."

In his Hunterian Lectures Prof. Alex. Hill says ('Brit. Med. Jrnl.,' Mar. 14, 1885):—"Within the grey matter we find three distinct kinds of cells: 1, large motor cells (measuring from 67 to 135μ) with distinct processes of Deiters, collected in two separate groups in the anterior and lateral horns respectively, except in the cervical and lumbar enlargements, where these groups fuse together; 2, a dorso-mesial group of cells (Clarke's column) resembling the cells of the anterior horn in form, but not in size, their diameters varying from 40 to 90μ; 3, spindle-shaped cells, of an average length of 18μ apparently devoid of an axis cylinder process. These cells are diffused through the matrix of the posterior horns."

"It must be remembered that the above-mentioned groups of cells constitute continuous columns, presenting nevertheless, a distinctly metameric arrangement. Birge has shown that in the frog, the number of motor cells in the spinal cord equals the number of large motor fibrils in the anterior roots leaving that metamer. Although there are certain difficulties in carrying out this investigation which should make us hesitate in accepting the exact numerical equivalence as proved, Birge's results are strongly confirmatory of the opinion that every motor fibre is connected with a nerve cell immediately before its exit from the cord. The variation in size of the posterior cornu, and consequently in the number of spindle-shaped cells which it contains, indicates that a similar connection obtains of the fibres of the posterior roots with nerve cells of their own metamer."

"Gaskell has, quite recently, called particular attention to the white rami communicantes (rami advehentes of Remak) of the sympathetic system, composed of the smallest medullated (leucenteric) fibres; and has further remarked that the number of cells in Clarke's column in any particular region appears to vary as the number of leucenteric fibres derived from that region. As the result, therefore, of Gaskell's researches, this column, hitherto so anomalous in position, in the character of its cells, and in its pathological alterations, falls into place as containing the primary centres of the visceral nerves; and we are justified in considering the central grey tube of the cerebro-spinal system as composed of metameric groups of three varieties of cells, well defined as to their histological characters, and severally connected with three kinds of nerve fibres, equally distinct as to their anatomical and their physiological relations."

Another view concerning Clarke's column is that its cells form the starting points for the fibres of the direct cerebellar tract—and for this notion good anatomical evidence can be adduced (p. 511). The two views, however, are decidedly incompatible. The small leucenteric fibres thought by Gaskell to be in relation with the cells of Clarke's column are said to be efferent fibres (see p. 519), while the fibres of the direct cerebellar tract are certainly afferent or centripetal fibres, which degenerate upwards (p 515).

In the medulla oblongata, "Clarke's column, the cells of which retain the same characters as in the cord, swells out into the nucleus of the vagus, the great leucenteric nerve of the thoracic viscera. From it also arise fibres of the glosso-pharyngeal nerve, which Vulpian has shown to exercise a vaso-dilator influence upon the back of the tongue; and into its anterior part Duval has traced the pars intermedia of Wrisberg, ramus visceralis of the seventh pair, from which the chorda tympani, vaso-dilator nerve of the submaxillary gland is derived. The sensory part of the glosso-pharyngeal nerve probably terminates in the spindle-shaped cells of the grey matter of the medulla."

(b).—Mode of Distribution of the Fibres of the Anterior and Posterior Roots in the Spinal Cord.

It is not needful to enter into any minute details concerning this difficult and much disputed subject. There is now a pretty general agreement concerning some of the modes in which these fibres are distributed, or at least as to the various directions in which they at first proceed. Our best present knowledge on this subject has been well illustrated by Schäfer in the following diagram (Fig. 118), to which we would direct the student's careful attention, as it embraces the principal points needful to be borne in mind.

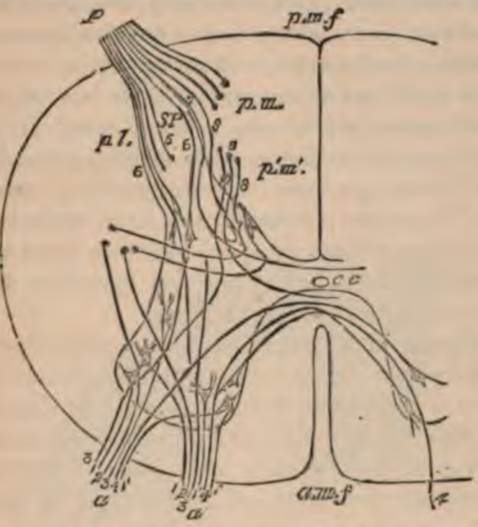

Fig. 118. Diagram to illustrate the course taken by the Fibres of the Nerve-Roots on entering the Spinal Cord [Schäfer].

a,*a*, Two funiculi of the anterior root of a nerve. 1, 1, Some of their fibres passing into the lateral cells of the anterior cornu. 1′,1′, Others passing into the mesial cells of the same cornu. 2, 2, Fibres passing to the lateral column of the same side without joining nerve-cells. 3, 3, Fibres passing towards the posterior cornu. 4, 4, Fibres passing across the anterior commissure to enter nerve cells in the anterior cornu of the other side.

p, Funiculus of the posterior root. *p*, *l*, Fibres of its lateral or external division coming through and around the gelatinous substance of Rolando: some of these, 5, are represented as becoming longitudinal in the latter; others, 6, 6, as passing towards the anterior cornu, either directly or after joining cells in the posterior cornu; and others, 7, as curving inwards towards the grey commissure. *p*, *m*, fibres of the mesial or inner division, entering the posterior column and then becoming longitudinal; *p*′, *m*′, fibres from a posterior root which had joined the cord lower down and entered the posterior column, now passing into the grey matter at the root of the posterior cornu. Of these, 8, is represented as entering Clarke's column; 9, as curving round this and coursing to the anterior commissure, and 10, as passing towards the anterior cornu. The axis-cylinder processes of the cells of Clarke's column are shown arching round, and taking the direction of the lateral column.

a.m.f., Anterior median fissure. *p.m.f.*, Posterior median fissure. *c.c.*, Central Canal. *s.R.*, Substantia gelatinosa of Rolando.

(c).—Fundamental Tracts into which the different Columns of the Cord are divisible.

Only such brief details will here be given as are absolutely needful to be borne in mind by those who seek to understand diseases of the spinal cord from the modern standpoint.

In no department of medicine have greater advances been made in recent years than in that relating to diseases of the part with which we are now concerned. Unquestionably the advances in clinical medicine that have been achieved in this direction have been owing, in the main, to the stimulus derived from several more or less concurrent investigations carried out by numerous workers from an anatomical, a developmental, and a physiological point of view; as well as from pathological researches having for their object to determine the course and extent of the various 'secondary degenerations' occurring within this organ. The results obtained by this latter method of investigation are of the most reliable order, and have been found to be in entire conformity with the more recent but very important developmental investigations made by Flechsig and others.

(1)—Developmental investigations have yielded valuable results of this sort. They have shown that the various strands of which the white columns of the cord are composed become developed at different periods, and that the different tracts are easily recognizable from one another, owing to the fact that when nerve fibres are first developed they are destitute of the white substance of Schwann; con-

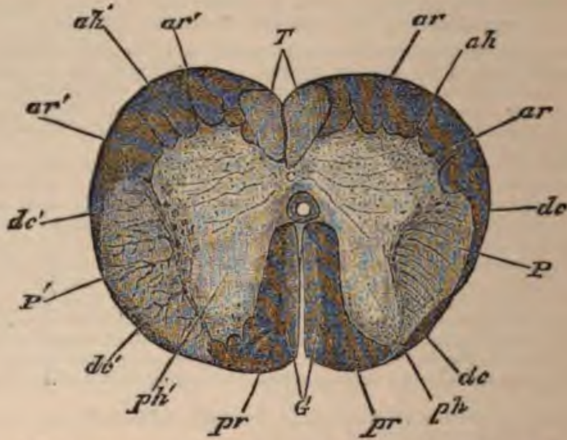

FIG. 119.—SECTION THROUGH THE CERVICAL REGION OF THE CORD OF A HUMAN EMBRYO AT THE FIFTH MONTH [after Ross].

ah, *ah'*, Anterior horns of grey matter.
ph, *ph'*, Posterior horns of grey matter.
ar, *ar'*, Anterior root-zones. *pr*, *pr*, Posterior root-zones.
P, P', Pyramidal fibres of lateral column.
T, Columns of Türck. G, Columns of Goll.
dc, *dc'*, Direct cerebellar fibres.
c, Anterior commissure.

sequently the sectional areas of the cord in which development is retarded remain of a greyish colour, contrasting notably with the dead white aspect of those whose development is more advanced, and in which the nerve fibres have already become surrounded by their white medullary sheaths. The tracts of imperfectly developed fibres, in fact, have an appearance to the naked eye, and also a mode of reacting to staining fluids, akin to that of the grey matter of the cord rather than to that of its fully developed white substance.

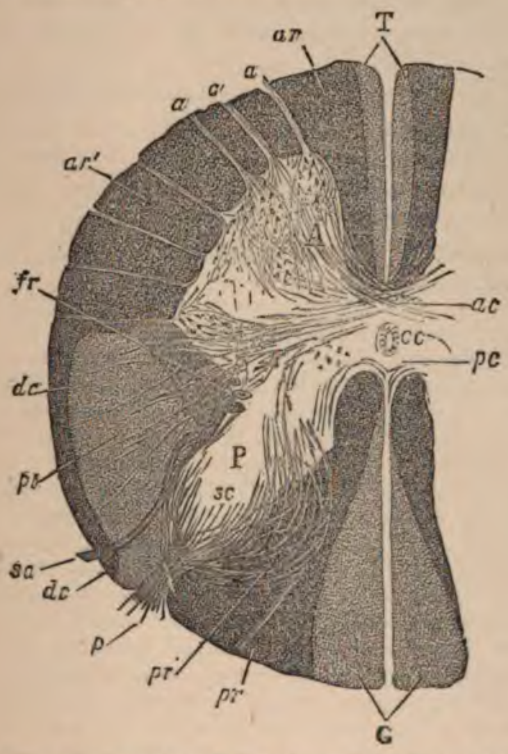

Fig. 120.—SECTION THROUGH THE SPINAL CORD OF A FŒTUS AT THE NINTH MONTH, ON A LEVEL WITH THE SECOND CERVICAL NERVE [after Ross].

T, Columns of Türck. G, Columns of Goll. *pt*, Pyramidal tract. *dc*, *dc*, Direct cerebellar tract.

A, P, Anterior and posterior grey cornua respectively. SG, Substantia gelatinosa. *cc*, Central canal.

ac, *pc*, Anterior and posterior commissure respectively.

p, Posterior root. *pr*, Posterior root-zone.

p'r, Internal radicular fasciculus.

a, *a*, *a*, Anterior roots. *ar*, *ar'*, Anterior root-zone.

fr, Formatio reticularis. *sa*, Spinal accessory nerve.

These differences in rate of development, and correlated differences in appearance, of different tracts of the spinal cord, cause the organ in the human subject to present a very striking aspect on transverse section, at any time during the latter half of fœtal life.

Thus, when the cord of a human embryo is examined at the end of the fifth month (Fig. 119), it will be found that the fibres in the pyramidal tracts of the lateral columns, the fibres of the columns of Türck, and also those of the columns of Goll, are non-medullated; while the fibres of the anterior and posterior root zones, and those of the direct cerebellar tracts in the lateral columns, are medullated.

At birth, the same tracts of the spinal cord still remain undeveloped, as may be seen from an examination of Fig. 120, which also represents a section through the cervical region. They are not, however, so undeveloped as they were, since at this period some of the fibres in these areas are found to have become medullated, though not in sufficient numbers to produce any notable alteration in the naked-eye appearance of the several tracts.

Ross ('The Diseases of the Nervous System,' Vol. II.), says:—"Inasmuch as the greater part of the fibres of the anterior and posterior root-zones, as well as those of the direct cerebellar tract are medullated as early as the fifth month of embryonic life, it may be presumed that all of them are fully developed at birth. The case, however, is different with regard to the fibres of the pyramidal tract, some of them being medullated and fully developed at the ninth month of embryonic life, while others are not. The fibres of the columns are probably also not all fully developed at birth. The fibres of the pyramidal tract in the cord are separated by the septa of neuroglia and the branching vessels into small lozenge-shaped spaces (Fig. 121). The later-formed fibres appear to insinuate themselves from above downwards along the margins of these spaces, so that the earlier formed fibres occupy their centres; the older being therefore further removed from the blood-vessels than the younger fibres. It may be assumed that the earlier-formed fibres connect the cortex of the brain with the earlier-formed or fundamental ganglion cells of the anterior horns, while the later-formed fibres connect the cortex with the accessory cells. What has already been said with regard to the size of the ganglion cells as a test of the stage of development of the cell is equally true with regard to the diameter of the medullated fibres. The diameter of these fibres may be accepted as a rough test of the age of the fibres during the period of development, but no longer. It is very probable that the small medullated fibres of the pyramidal tract connect together the small cells of the anterior horns and relatively small cells in the cortex of the brain; while on the contrary the thick fibres connect the large ganglion cells of the anterior horns and large cells of the cortex. The largest cells of the spinal cord, for instance, are found in the lumbar region, and the largest in the cortex of the brain in the paracentral lobule—the centre of the movements of the leg—and it is probable that these cells are connected with each other by thick fibres. We have already seen that, as a rule, the accessory are smaller than the fundamental ganglion cells of the anterior horns, and it may therefore be inferred that the accessory fibres of the pyramidal tract are, as a rule, smaller than the fundamental ones. The smaller fibres are found in greater numbers in the internal and posterior parts of the lateral columns, the portion of the white column which adjoins the grey substance. At this spot the septa of connective tissue are larger, the neuroglia is more spongy, and the lozenge-shaped spaces already described are more distinctly marked than in the more external layers of the white substance."

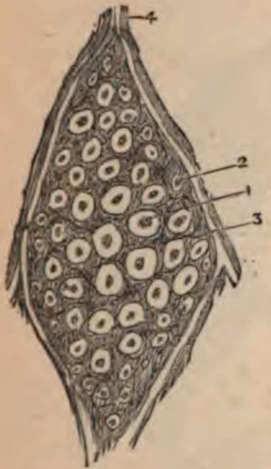

FIG. 121.—TRANSVERSE SECTION OF A PORTION OF THE PYRAMIDAL TRACT, MAGNIFIED [after Ross].

1, Fibres of large diameter. 2, Fibres of small diameter. 3, Deiter's cells. 4, Twig of the median branch of the central artery of the spinal cord.

The various developmental observations on the spinal cord will be found to yield results closely agreeing with those which had long ago been arrived at from the study of ascending and descending 'secondary degenerations' in the spinal cord. These latter data will presently be referred to more in detail; meanwhile it may be well to note the definite tracts or fasciculi into which the white substance of the cord may be divided as a result of Flechsig's developmental observations. These are shown in Fig. 122, from which it may be seen that the posterior column is divided into two distinct tracts, viz., the column of Goll, and the posterior root-zone; while the antero-lateral column is at least divisible into four readily distinguishable tracts, viz., the crossed pyramidal tract, the direct cerebellar tract, the column of Türck, and the very large intermediate area known as the anterior root-zone or 'mixed tract.'

Some further details are needed concerning the connections, the longitudinal extent, and the relative size of these different columns at different levels of the cord.

The results obtained by Flechsig in this direction may be best understood by the examination of Fig. 123, accompanied by some brief explanations.

These diagrams represent sections of the cord at different levels;

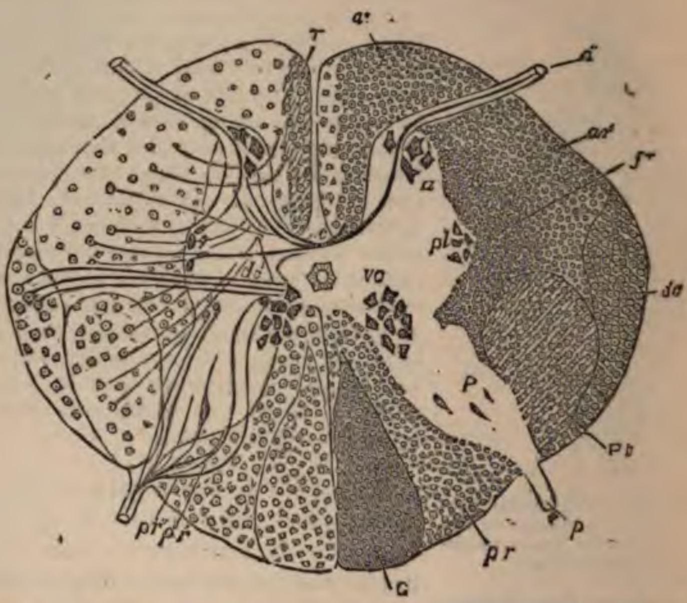

FIG. 122. DIAGRAM OF A TRANSVERSE SECTION OF THE SPINAL CORD IN THE UPPER HALF OF THE DORSAL REGION [after Flechsig].

Pt, Pyramidal tract of the lateral column.
T, Columns of Türck. *dc*, Direct Cerebellar tract.
ar, Internal portion of the anterior root-zone.
ar', External portion of the anterior root-zone.
pr, Posterior root-zone. G, Columns of Goll.
a, Anterior grey horns of the spinal cord.
c, Anterior commissure.
a', Fibres of anterior roots. *p*, Fibres of posterior roots.
vc, Vesicular column of Clarke.
dc', Fibres which pass from the vesicular column of Clarke to the direct cerebellar tract.
fr, Reticular formation of the spinal cord.

and they show that the number, the exact disposition, and the relative size of the different tracts into which the columns of the cord are divisible are subject to definite variations at different levels.

These different tracts are similarly numbered in the several sections, in accordance with their relative priority in the commencement or attainment of full development. A few words may be said concerning each, in this same order.

1, Is known as the principal mass of the anterior column, representing all except about the inner fourth of this column. It possesses about the same relative size at different levels of the cord. It

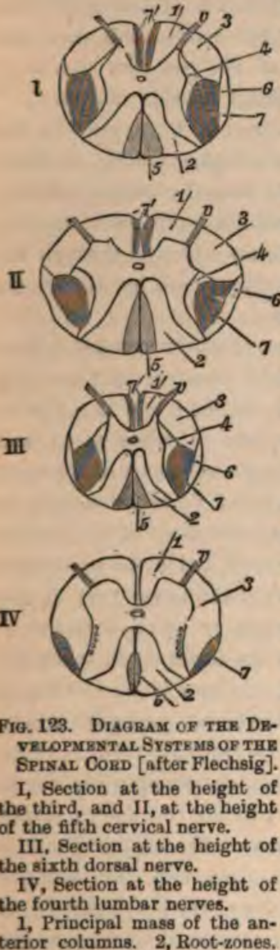

FIG. 123. DIAGRAM OF THE DEVELOPMENTAL SYSTEMS OF THE SPINAL CORD [after Flechsig].

I, Section at the height of the third, and II, at the height of the fifth cervical nerve.

III, Section at the height of the sixth dorsal nerve.

IV, Section at the height of the fourth lumbar nerves.

1, Principal mass of the anterior columns. 2, Root-zones. 3, Anterior mixed region of the lateral columns. 4, Lateral boundary layer of the grey substance. 5, Columns of Goll. 6, Direct Cerebellar tracts. 7, pyramidal tracts of the lateral columns. 7′, Pyramidal tracts of the anterior columns. *v*, Anterior roots.

is composed in part of fibres proceeding to the anterior roots, and in part, it is supposed to consist of short commissural fibres. Our knowledge concerning these tracts is still very vague.

2, Is the so-called root-zone (or wedge tract), corresponding with the posterior column exclusive of the column of Goll. This tract varies greatly in bulk in different parts of the cord, being largest in the situation of the cervical and lumbar swellings. It is composed in the main of direct continuations of the posterior roots, though amongst them there are numerous bundles of longitudinal fibres.

3, Is the anterior half of the lateral column, known as the anterior mixed region of the lateral column. In part the fibres of this region are the direct continuations of the anterior roots, and in part they are composed of short commissural fibres; much more information is, however, needed in regard to them.

4, Is known as the lateral boundary layer of the grey matter. Nothing definite is known concerning the longitudinal fibres in this situation—as to their origin, their destination, or their functions. It is known, however, that this tract is crossed more or less horizontally by two distinct sets of fibres, (*a*) fibres of the crossed pyramidal tracts, and (*b*) fibres passing from the ganglion cells of Clarke's column outwards to ascend in the direct cerebellar tract (Fig. 122, *dc′*).

5, Is the so-called column of Goll, which is very small in the lumbar region, but gradually increases in bulk upwards through the dorsal and cervical regions of the cord. It is made up of fibres which seem to terminate in the cuneate nucleus of the medulla oblongata, while below they come from successive segments of the cord from the lumbar region upwards. At their lower extremities they are probably in relation with ganglion cells of the posterior cornu of the cord, though we have no definite knowledge on this subject.

6, The direct cerebellar tract begins to appear in the upper part of the lumbar enlargement, as a compact bundle of fibres at the periphery of the posterior half of the lateral column just outside the crossed pyramidal tract. This bundle increases in size, by the constant accession of new fibres, as it passes upwards through the dorsal and cervical regions of the cord. Its fibres pass by way of the restiform body to the cerebellum (see p. 517).

7, Is the crossed pyramidal tract, whose general course from above downwards we have already had occasion to trace (pp. 202-216). If we examine it from below, it will be found to make its appearance first in the lower half of the lumbar enlargement, and gradually to increase in sectional area in higher segments of the cord, as it approaches nearer and nearer to its point of decussation with its fellow in the lower part of the bulb. Throughout most of the lumbar region, its fibres reach the periphery of the cord, but in the upper part of the lumbar region, in the dorsal, and also in the lower part of the cervical region, it is separated from the periphery by the direct cerebellar tract. In the upper cervical region its fibres again touch the periphery posteriorly. Much variation is at times found to exist in the size of the crossed as compared with the direct pyramidal tracts. This subject has been elsewhere referred to (pp. 161-163). Occasionally a very unsymmetrical arrangement exists, which gives rise to marked and quite obvious lack of symmetry in the two halves of the cord, due to the fact that by far the larger quantity of the fibres of one anterior pyramid of the medulla may pass into the opposite lateral column, whilst those of the others may scarcely decussate at all, but pass, in the main, along the anterior column of the cord on the same side.

7′, Is the direct pyramidal tract of the anterior column. The lowest level at which these tracts show themselves, is subject to much variation in different individuals. Sometimes it is in the lower part of the lumbar enlargement; at other times it is in the upper part of this region; though more commonly still it is in the lower part of the dorsal region that these tracts begin to make their appearance. They gradually increase in size upwards, and from what has been said in reference to the crossed pyramidal tracts, it will follow that their relative size, compared with these latter columns, is also subject to much variation in different individuals.

(2).—The study of **Secondary Degenerations in the Spinal Cord,** some thirty years ago, was, as we have already stated, the first mode in which a knowledge was obtained (especially through the investigations of Türck), that the white columns of the spinal cord were divisible into certain definite secondary tracts. The results of the earlier researches concerning secondary degenerations have been substantiated by the later developmental studies of Flechsig and others, though they have not been notably extended thereby.

The study of secondary degenerations, moreover, gives us a kind of information which is not supplied by developmental investigations. It tells us which sets of fibres have efferent, and which of them have afferent, functions. From the first, secondary degenerations in the spinal cord have been known as 'descending' and 'ascending degenerations,' and it has always been distinctly understood (upon the basis of Waller's experiments) that descending degenerations involve tracts of efferent fibres; and, on the other hand, that ascending degenerations occur in tracts of afferent fibres.

Little requires to be said on this subject here, since it has been in part discussed in connection with Paralyses of Encephalic Origin (p. 197); while, moreover, the relative size and distribution of the tracts of secondary degeneration in the cord have, in effect, been already pretty accurately described in the previous section, in which the results of Flechsig's developmental observations have been set forth.

Descending degenerations, especially, may be dismissed in a very few words. These may occur in four situations in the spinal cord, viz., in the 'direct' and in the 'crossed pyramidal tract' of each side.

Such degenerations are met with, in the main, in association with two sets of conditions :—

(*a*). They occur in association with hemiplegia, when this is due to a unilateral brain lesion which has more or less damaged the opposite pyramidal tract in some parts of its course between the cortex and the bulb. In such a case the 'crossed pyramidal tract,' in the spinal cord, is degenerated on the paralysed side (Fig. 124, A), while the 'direct pyramidal tract,' shows the same kind of change on the side of the brain lesion.

(*b*). They occur also in association with paraplegia, and in

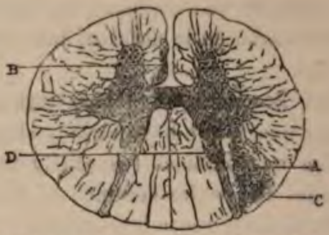

FIG. 124. DESCENDING SECONDARY DEGENERATIONS IN THE SPINAL CORD, COEXISTING WITH A RIGHT SIDED HEMIPLEGIA [after Charcot].

Transverse section of the cord in the cervical region. A, degeneration of the crossed pyramidal tract in the right lateral column. B, degeneration of the direct pyramidal tract. C, area of white matter corresponding to the direct cerebellar tract. D, region intermediate between the posterior cornu and the pyramidal tract, which is never affected in descending degeneration.

their most typical form when either injury or disease has disorganized the whole thickness of the cord, somewhere in the cervical or in the upper dorsal region. In such a case degeneration occurs below the seat of lesion, throughout both 'crossed' and both 'direct pyramidal tracts,' the areas of degeneration wearing themselves out below in the manner which has been described for the pyramidal tracts themselves.

Speaking generally, it may be said that the anterior areas of degeneration in such cases are apt to be larger and better defined than when they occur in association with hemiplegia, as a result of a brain lesion. Of course the relative size of the areas of degeneration in the anterior and in the lateral columns respectively, is liable to vary in different individuals in accordance with the varying degrees of completeness in the decussation of the anterior pyramids (p. 161). In one case, observed many years ago, I found the extent of the degeneration in the anterior columns altogether remarkable (Fig. 125), and the areas in the lateral columns, moreover, notably

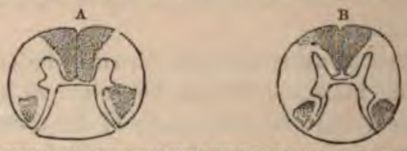

FIG. 125. DESCENDING DEGENERATIONS MET WITH IN A CASE OF CONCUSSION-LESION OF THE SPINAL CORD, IN THE CERVICAL REGION.

A. Section of the Cord just below the cervical enlargement.
B. Section through the mid-dorsal region of the Cord.

unequal. This latter point may have been due to the fact that the cord was not completely cut across or disorganized in the left lateral column at the seat of injury in the cervical region, so that the amount of degeneration was decidedly less on that side.

Below the seat of lesion, descending degenerations are apt also to exist in different parts of the 'mixed tracts,' but to extend only for a very short distance downwards. These are probably due to degenerations implicating 'short commissural fibres,' which, as Bouchard long ago indicated, seem to exist in this situation.

Ascending Degenerations in the spinal cord also occur in four principal regions, viz., in both columns of Goll, and at the periphery of the cord on each side, in the postero-lateral region. There is good reason, however, for believing that these latter tracts are each divisible into two, and in this case, there would really be six regions in the cord in which ascending degenerations occur.

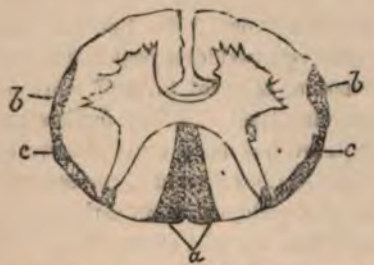

FIG. 126. ASCENDING DEGENERATIONS IN THE CERVICAL REGION OF THE CORD RESULTING FROM A DISORGANIZATION OF THE WHOLE THICKNESS OF THE CORD IN THE UPPER DORSAL REGION.

a, Degeneration in the columns of Goll. *c*, Direct Cerebellar tract. *b*, Distinct afferent tracts, supposed to conduct impressions of pain (lateral sensory tract).

In regard to the beginning and ending of the columns of Goll, nothing need be added to what has been said on p. 511 in reference to Flechsig's developmental region, No. 5. Nothing definite is known as to the functions subserved by the fibres of these columns.

The ascending degeneration in the lateral column of the cord was formerly imagined to belong entirely to the 'direct cerebellar tract,' whose mode of commencement and ending is also referred to on p. 512. The supposed connection of the fibres of these tracts with the cells of Clarke's column, as afferent fibres, must also be borne in mind (see Fig. 122).

There is now, however, reason to believe that the anterior, and often wedge-shaped, extremity of the tract of degeneration in the lateral column, which the writer figured in 1867 (Fig. 128, A), and which has been commonly regarded as forming part of the cerebellar tract, is, in reality, composed of an entirely distinct set of fibres. Our recognition of this is due to the fact that Gowers chanced to examine a cord in which a crushing lesion had occurred low down—too low down, that is, to give rise to degenerations in the 'direct cerebellar tracts.' Consequently, he found ('Diagnos. of Diseases of the Spinal Cord,' 1st Ed., 1880, p. 13) well-marked ascending degenerations in the columns of Goll, and also "a symmetrical area of slight ascending degeneration in the anterior part of the lateral columns" (Plate, Fig. 3, *e*). Gowers fancied that this was an altogether new area of degeneration. I could not share in this opinion, for as H. A. Tooth has recently pointed out ('St. Barthol. Hosp. Reports,' Vol. xxi.) several other observers besides myself had figured areas of degeneration in this situation, adjacent to, and more or less continuous with, the direct cerebellar tract. Tooth further says, in reference to this tract, that Bechterew has made some observations concerning its development. "He describes a bundle, corresponding in situation to the one in question, as developing at an earlier period than the pyramidal tract, and later than the rest of the lateral column. Bechterew considers that the fibres of this bundle are sensory and give passage to pain sensations. He does not appear to have known of Dr. Gowers' observation." The latter observer had also given reasons for supposing this tract to conduct sensory impressions of some kind. Perhaps it would be well, for the present, at all events, that we should designate it as the sensory tract of the lateral column, or, more briefly, the lateral sensory tract.

My own observation leads me to believe that considerable variation often exists in regard to the exact disposition of this lateral sensory tract and of the direct cerebellar tract, as well as in reference to their exact relations to one another. This is illustrated by the

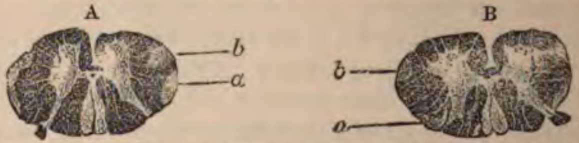

FIG. 127. ASCENDING DEGENERATIONS IN THE CERVICAL REGION OF THE SPINAL CORD, DEPENDENT UPON A CRUSHING LESION IN THE MID-DORSAL REGION [after H. Tooth].

A, Section of the Cord between the roots of the second and third cervical nerves.
B, Section of the Cord between the roots of the third and fourth cervical nerves.
a, Direct cerebellar tract. *b*, Lateral sensory tract.

unsymmetrical condition of the combined areas on the two sides of Fig. 126, and by the much more notable variations in two sections depicted by Dr. Tooth in illustration of his paper (Fig. 127). Here the disposition of the tracts is dissimilar in the two sections, and altogether unsymmetrical on the two sides, in each of them.

Again, in the case which I described in 1867, there was a remarkable disposition of the tracts of degeneration in the posterior columns (Fig. 128) such as I have never seen since, either actually in spinal cords or represented in books. Unfortunately, in this particular case, the exact nature and extent of the lesion in the lower cervical region of the cord which gave rise to the ascending degenerations was not ascertained. The tract of degeneration in the lateral column was distinctly traced by me into the restiform bodies.

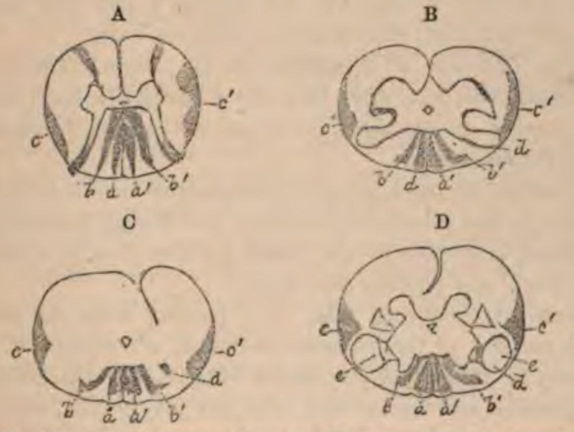

FIG. 128. PECULIAR AREAS OF ASCENDING DEGENERATION, MET WITH IN A CONCUSSION-LESION OF THE SPINAL CORD.

A. Transverse section of the spinal cord, near the middle of the cervical region.
B. Transverse section through the cervical region of the cord, near lower border of medulla.
C. Section through lower part of medulla oblongata.
D. Section through medulla about ¼″ below the *Calamus Scriptorius*.

The areas in the posterior columns (*a*, *a′*, *b*, *b′*) have a very peculiar disposition. The areas in the lateral columns in A are very unsymmetrical, that of the right side, anterior to *c′*, represents the lateral sensory tract.

Higher sections through the Medulla are represented in *Med. Chir. Trans.*, 1867, Pl. IX.

It should also be stated that, for a short distance above an injury or lesion which cuts across the whole of the fibres of the posterior columns, or the whole thickness of the cord, ascending degenerations are found in the root-zones or postero-external columns of the cord. These degenerations probably occur in short commissural fibres in this situation, or in fibres derived from the posterior roots which, after a short upward course, terminate in the posterior grey cornua. This fact, however, throws no light upon the remarkable disposition of the ascending areas of degeneration in the posterior columns of the case above referred to. Here we had tracts partly occupying the postero-internal and partly the postero-external columns, and just as well defined in the one as in the other.

(d).—Nature of the Functions subserved by the various Columns and parts of the Grey Matter of the Spinal Cord.

Our knowledge in regard to this subject is still very incomplete. Only a few brief hints will, therefore, be given here.

In-going Channels of Conduction to the Brain.—The paths for these impressions, soon after their entry into the spinal cord by the posterior roots, cross to the opposite half of the cord, decussating with those of the other side.

Impressions of touch, pressure, temperature, and tickling travel in the main by the posterior columns—though probably some of them ascend in portions of the lateral columns, that is, in the 'lateral sensory tract' and perhaps elsewhere.

Impressions producing pain, after passing with the nerve-roots through portions of the posterior columns, are commonly supposed principally to traverse the grey matter of the cord. Disease or damage of the posterior columns, as well as of the grey matter, often causes a more or less marked retardation in the transmission of such impressions. Bechterew thinks that impressions producing pain, pass upwards through the 'lateral sensory tracts.'

The path traversed by impressions from muscles to the encephalon (so important for the regulation of movements) is not distinctly known —but it is possibly to be found in the posterior columns. These channels, moreover, are said by Brown-Séquard to decussate in the pons Varolii, rather than soon after their entry into the spinal cord.

The path for the transmission of impressions from the 'genital centres,' in the lumbar region of the cord, to the brain, is also probably situated in the posterior columns.

The physiological anatomy of the cord in the lumbar region seems to be slightly different from what it is higher up in the dorsal and cervical regions. In the former situation, ordinary sensory impressions from the lower extremities are said to pass through the lateral, rather than through the posterior columns. Those from the pelvic region, the sexual organs, the perinæum and anal regions, however, are thought to pass upwards through the posterior columns.

It is doubtful what kind of impressions pass up through the direct cerebellar tract. It is generally supposed that these fibres emanate below from the cells of Clarke's column. On the other hand, Gaskell ('Jrnl. of Physiol.,' Vol. vii, No. 1, pp. 56 and 67) is strongly of opinion that these cells are in relation with efferent vaso-motor and visceral fibres, and doubts their connection with afferent fibres. There appears to be no possibility of reconciling these views, at present. It seems certain that the cerebellum receives a large body of afferent impressions through the spinal cord (see p. 225). The only route for such impressions of which we now have any knowledge is the 'direct cerebellar tract.' There may, however, be others, and it is possible that the columns of Goll may be parts having an important function of this kind.

Out-going Channels of Conduction from the Brain.—All that is certainly known concerning the paths for voluntary motor incitations is that, below the decussation of the pyramids, they are to be found mainly in the posterior part of the lateral column ('crossed pyramidal tract'). The fibres descend through these columns to different levels, according as their stimuli are destined to evoke the activity of different nerves and muscles, as already explained (p. 211).

The motor paths for the foot and leg, in the lumbar lateral column, are said to lie more towards the circumference of the cord than those for the thigh-muscles.

On the other hand, some of the pyramidal fibres of the medulla pass into and through the spinal cord on the same side, that is, without decussating, so as to constitute the inner part of the anterior columns ('direct pyramidal tract'). Some of its fibres are thought to cross into the anterior horn of the opposite side, by means of the anterior or white commissure. What is the precise function of these fibres in relation to those of the 'crossed pyramidal tract,' is not as yet known. The only common point of agreement is, that these fibres of the 'direct pyramidal tract' are in some way concerned with movement rather than with sensation. It has been supposed by some that they are fibres whose decussation takes place at different sites below the medulla.

It seems also worthy of consideration whether they may not be efferent fibres for the conduction of cerebellar influence to the grey matter of the spinal cord (see p. 222). It is almost certain that some such efferent fibres exist in the spinal cord though they have not as yet been identified or localised in any way.

It is probably an error to suppose that there are any special routes for the conduction of reflex motor impulses from the brain, apart from those concerned with the excitation of voluntary movements.

Some speculations as to the functions with which different columns of ganglion cells in the grey matter of the spinal cord are related, have been already referred to (pp. 517-521).

REGIONAL DIAGNOSIS.

GENERAL CONSIDERATIONS RELATING TO THE REGIONAL DIAGNOSIS IN PARALYSES OF SPINAL ORIGIN.

Preliminary Remarks.

For reasons which we have already pointed out (p. 6), it is on the whole more convenient in dealing with paralyses of spinal origin to enter upon the consideration of the regional diagnosis before, rather than after, the general consideration of the pathological diagnosis.

Although it is true that the groups of symptoms presented in different diseases of the spinal cord, considered individually and collectively, afford the materials upon which a regional diagnosis must be founded, it is no less true that a part of the symptomatology (namely, that comprised in the mode of origin and the mode of establishment of the disease, together with what may be gathered from the patient's state generally, from his family history, and from his personal history) constitutes the basis upon which a pathological diagnosis has to be arrived at.

Again, although the arrival at a regional diagnosis is often spoken of, and may seem to be a process altogether distinct from that involved in the arrival at a pathological diagnosis, yet, as a matter of fact, in the investigation of many individual cases of spinal disease, it will be found that the one problem is not settled first, and the other afterwards, but that both are tentatively considered more or less simultaneously. Thus, certain empirically known pathological conditions may afford at once a ready explanation of a given group or sequence of symptoms, as in 'infantile paralysis,' in 'locomotor ataxy,' or, in a more general sense, in angular curvature of the spine. Here, therefore, the pathological diagnosis goes hand in hand with the regional diagnosis, and in working them out each gathers additional confirmation from the establishment of the other. Sometimes, however, as in the case of traumatic injuries (including stabs, and fractures with dislocations of vertebræ), the pathological diagnosis is at once obvious, and the regional diagnosis alone requires to be settled in detail.

Preliminary to the consideration of Paralyses of Encephalic Origin, it was deemed expedient to enter upon a rather long discussion of the "Regional or Localising Value of the Special Signs which may be associated with such Paralyses." Here, however, by reason of the lesser complicacy of the phenomena associated with diseases of the spinal cord, no such preliminary discussion is needful. It will be found that all the essential facts are referred to in one or other of the next two sections.

CLINICAL INDICATIONS FAVOURING THE DIAGNOSIS OF DISEASE IN THIS OR THAT REGION OF THE SPINAL CORD.

The regional diagnosis of diseases of the spinal cord is itself a twofold problem. It involves a consideration : (*a*) of the particular part of the transverse area of the spinal cord which is the seat of lesion; and (*b*) of the longitudinal situation and extent of the disease in such transverse areas.

I.—Diagnosis of the particular part of the transverse area of the Spinal Cord which is the "seat" of Lesion.

The facts to be tabulated under this head may be set down in the order of their relation to different component parts or regions of the spinal cord.

(1) *Anterior Roots of Spinal Nerves.*—Irritation of these may give rise to various forms of twitching, or to tonic spasms in related muscles. Great pressure upon, or destruction of, the anterior roots will give rise to local paralysis in the functionally related muscles; followed, in the course of a week or two, by marked atrophy, and the establishment of the electrical 'reaction of degeneration' (see p. 10). There will also be an abolition of the reflex excitability of these muscles in response to skin-irritation, or from blows upon or stretchings of their tendons.

(2) *Antero-lateral Columns.*—Increasing pressure upon or disease of these columns gives rise to paresis, gradually deepening into motor paralysis, of parts deriving their nerve-supply at or below the seat of lesion.

When the disease occurs in the lateral column more especially, there may be twitchings or startings in the muscles below, or well-marked spasms, and possibly painful cramps. There may also be great exaltation of the superficial and deep reflexes, if the manifestation of the latter be not hindered by pre-existing spasms. Motor paralysis exists to some extent, but without any very appreciable impairment of sensibility. No marked wasting of muscles, or diminution of their electrical reactions, usually occurs.

(3) *Grey matter.*—*a*, Of anterior Cornua.—Disease of these parts causes motor paralysis, atrophy, loss of faradic excitability, and loss of reflex excitability in related muscles—as in cases of disease of the anterior roots of spinal nerves.

b, Of posterior Cornua and Central Parts.—Damage of these regions of grey matter will, according to its completeness in transverse extent, cause more or less delay or defect in the transmission of painful impressions, and perhaps interfere also with other modes of sensibility.

Some trophic lesions in skin and joints, which are referred to below, may also be met with.

At different levels in the cord special centres (represented in both anterior and posterior regions of grey matter) in connection with definite functions, may be interfered with by morbid conditions implicating the grey matter (see p. 526).

(4) *Posterior columns.*—The results of disease confined to this situation (more especially to the 'root-zones') will be ataxy or signs of incoordination of movements; interference with impressions of touch, pressure, temperature, and of 'muscular sense'; abolition of knee-reflex; and diminution or loss of sexual desire.

(5) *Posterior roots of spinal nerves.*—From irritating lesions there will arise lancinating or other pains in the skin and deeper textures of related portions of the limbs, and possibly trophic skin-lesions. Pressure or destructive lesions will give rise to loss, in various degrees, of different modes of sensibility, superficial and deep; as well as to diminution or abolition of the superficial and deep reflexes in related regions of the body.

Trophic Relations between different Tissues and different parts in the transverse area of the Spinal Cord.—Irritation of the posterior cornua, or of the posterior roots of the spinal nerves, may give rise to various pustular or vesicular eruptions in related portions of the skin, often associated with neuralgic pains in these same regions. In other cases, with lesions in some parts of the grey matter, ulceration or actual sloughing of certain related tracts of skin are easily determined—especially under the combined influence of continued external pressure, and frequent irritation from urine or fæces, as in some cases of paraplegia.

Degeneration or destruction from any cause of the great ganglion-cells of the anterior cornua, or of the anterior roots of the spinal nerves (either within or outside the cord), gives rise, in the course of two or three weeks, to atrophy of the muscle-fibres with which such cells or nerve-roots are in relation. We thus get an atrophic paralysis, associated with the electrical 'reaction of degeneration' (p. 10).

Certain diseases affecting the grey matter or posterior roots (in ways and sites that cannot be precisely defined) are also apt to be associated with chronic diseases of joints. Sometimes comparatively unimportant, they lead, in other instances, to great atrophy of the articular ends of the bones, and possibly to dislocation with utter destruction of the joint, as in some cases of locomotor ataxy (Charcot's arthropathy). Atrophy, with brittleness of bones, may also be met with in similar or in allied cases.

The fact of the existence of these trophic troubles in association with such lesions, may be admitted wholly irrespective of the explanation of their pathogenesis. Whether they are due to altered states or influences transmitted to the respective tissues by the ordinary motor and sensory nerves in relation with such tissues, or to altered influences emanating from certain purely hypothetical 'trophic nerves,' lies altogether outside the fact of the mere coexistence of the several trophic troubles with the several lesions—which is the point of more immediate interest for the practitioner of medicine.

II.—Diagnosis of the Longitudinal Situation and Extent of the Lesion.

This is a consideration distinctly secondary to the other, since at whatever longitudinal level the disease may be situated, its clinical characters will always be qualified by reason of the parts of the transverse area of the cord that may be involved. For the determination of the point with which we are now concerned, dependence must be placed, in the main, upon signs indicative of the implication of particular sensory and motor nerves, whose exact relations with different longitudinal levels of the spinal cord are, of course, known. Such signs may consist of some excess or defect of sensibility, of motility, or of reflex action.

We are accustomed also to obtain information of a more general kind from the fact that this or that special centre, in connection with different viscera and functions, situated at different longitudinal levels in the cord, may be deranged in this or that case, and thus give an indication as to the height in the cord to which the lesion or damage extends. To this latter portion of the subject it will be found most convenient to give attention in the first place.

(*a*). *Evidence as to the Longitudinal Situation of the Lesion, derivable from Perversions of Activity of different Spinal Centres.*

(1) The lateral columns in the upper cervical region contain the motor paths for the muscles of respiration, so that section or disease of these columns at a lower level interferes with the movements of respiration on the same side of the chest (thoracic muscles); while, if the lesion reaches as high as the fourth and third cervical nerves (the origin of the phrenic), the diaphragm itself also becomes paralysed, and the movements of respiration must therefore almost cease.

(2) Again, the upper cervical region of the cord, if it does not contain actual centres connected with the excitation of the heart's action, is at all events traversed by the augmentor and accelerator nerves whose point of exit from the cord, as white rami viscerales, is with the 2nd, 3rd and other upper dorsal anterior roots. These nerves ascend in the sympathetic to the stellate ganglion, and thence proceed to the heart.

Thus, different lesions in this upper cervical region of the cord may, according to their nature and extent, greatly interfere with the heart's action, as well as with the respiratory movements. The frequency of the pulse may be either notably accelerated or retarded, according as the lesion is of an irritative or destructive nature; while the respiratory movements may be slower or much quicker than natural, or they may be extremely irregular and perverted in rhythm.

(3) The lower cervical and upper dorsal regions of the cord (that is, as low as the level of the second dorsal nerve), also contain the so-called 'cilio-spinal centre', or the fibres emanating therefrom. These pass outwards with the fibres of the anterior roots in the above-named regions, and thence into the cervical sympathetic. Irritation of them causes dilatation, together with sluggish action, of the pupil on the same side; widening of the palpebral fissure; prominence of the eyeball associated with a feeling of tension (as in glau-

coma), and a scanty secretion of tears and mucus. Section or other destructive lesion of these fibres, on the other hand, causes contraction of the pupil and other signs the reverse of those produced by irritation.

(4) The vaso-motor nerves for the side of the head and neck issue from the spinal cord with the anterior roots of the second, third and fourth dorsal nerves, thence ascending in the cervical sympathetic to their destination. Irritation of them produces contraction of the blood-vessels and a lowering of temperature of the side of the face and head; together with diminution of sensibility, an absence of perspiration, and (should the irritation continue) a tendency to slight atrophy of the side of the face. Destructive lesions operating upon these vaso-motor fibres, tend to produce a reverse set of conditions.

Sometimes there may be signs of paralysis of oculo-pupillary fibres coexisting with signs of irritation of the vaso-motor fibres, or *vice-versâ.*

(5) Gaskell has shown ('Jrnl. of Physiol.,' Vol. VII.) that all the vaso-motor nerves of the body leave the spinal cord in the anterior roots of the spinal nerves, from the second dorsal to the second lumbar inclusive, passing thence into the lateral ganglia of the sympathetic, in which these vaso-motor fibres lose their medullary sheaths.

He thinks that the vaso-motor nerves are (in common with some ordinary motor nerves) connected with the lateral horns of the spinal cord. He writes as follows (*loc. cit.*, p. 59) :—" The experiments upon the position of the vaso-motor centre in Ludwig's laboratory, seem to show that the vaso-motor nerves not only pass along the cord from the medulla oblongata in lateral tracts, but also are in close connection with a group of nerve cells in the medulla, known as the antero-lateral nucleus of Clarke. This group of nerve cells is apparently an isolated portion of the cell column of the lateral horn which has been cut off by the crossing of the pyramids. It is, therefore, possible that the cell column of the lateral horn has a connection with vaso-motor nerves as well as with ordinary motor nerves. In this case we must look upon the ramus visceralis as arising not wholly from the cells of Clarke's column, but also from those of the lateral horn."

According to Gaskell, the outflowing streams of vaso-motor nerves from the thoracic region of the cord come into direct relation with the lateral sympathetic ganglia, and leave them as non-modulated fibres. He adds (*loc. cit.*, p. 15) :—"This chain might, therefore, most appropriately be called the chain of vaso-motor ganglia, instead of its present meaningless title of main sympathetic chain."

According to Cyon the vaso-constrictor nerves for the upper extremity pass out of the cord (below the origin of the roots of the brachial nerves) in the anterior roots of the second and subsequent thoracic nerves, and reach the brachial plexus by way of the ganglion stellatum.

On the other hand, Heidenhain and Ostroumoff have shown that the vaso-constrictor nerves for the foot leave the spinal cord to reach the sympathetic, high above the origin of the roots of the sciatic (viz., from the lower dorsal and upper two lumbar roots), and that they reach the sciatic nerves by way of the abdominal sympathetic chain. The vaso-dilator nerves are, however, supposed to pass out from the spinal cord in the roots of the sciatic nerves—and, according to Stricker, they are to be found in their posterior roots.

It is generally thought that section of one half of the cord, or destruction of it for any extent longitudinally, causes at first paralysis of blood-vessels in the lower parts of the body on the same side—this vaso-motor paralysis carrying with it, in the same parts, an increase of temperature and an exaltation of sensibility. In a short time, however, the vaso-motor paralysis (and with it the increase of heat and sensibility) passes away, owing to the vaso-motor centres in parts of the spinal cord below, and to the peripheral vaso-motor centres, adapting themselves to act independently of those in higher parts of the cord and of the supreme regulating centre in the medulla oblongata. (As a rule the higher vaso-motor centres control those lower down, but after paralysis of one of these higher centres even the peripheral vaso-motor centres seem to be capable of resuming control over related blood-vessels.)

(6) The movements of the stomach and intestines generally are certainly influenced by the cord in different regions, so that in various cases, under perversions of this normal spinal influence, we may get vomiting, diarrhœa, or obstinate constipation—as direct results, that is, of morbid changes in certain parts of the cord in which intestinal pneumo-gastric, or sympathetic, fibres have their roots. The exact situations of these centres and paths of stimulation are, however, only vaguely known—epecially those which supply the longitudinal muscles of the alimentary canal.

Gaskell says (*loc. cit.*, p. 18) :—"We see at the outset that the nerves of the alimentary canal and its appendages can be divided into two groups, those which supply the longitudinal and circular muscles respectively; of these two divisions it is well known that throughout the greater portion of the alimentary tract the vagus supplies the motor fibres for the latter layer of muscles, causing when stimulated the well-known peristaltic movements. Such peristaltic contractions of the œsophagus, stomach, and intestines can be excited by vagus stimulation, whether the stimulus be applied to the roots of the nerve as they leave the medulla, or to the main trunk in any part of its cervical or thoracic course. Such nerves, then, must form part of the cervico-cranial outflow of visceral nerves and leave the central nervous system in the roots of such nerves as the accessory, vagus, and glosso-pharyngeal." In these roots they are medullated fibres, but, beyond the ganglion of the trunk of the vagus, they pass to their distribution as non-medullated fibres.

While the fibres for the circular fibres of the greater part of the alimentary canal are connected only with the upper cervical region of the cord, it is important to remember that the circular fibres of the rectum are supplied by the lower abdominal splanchnic nerves, which leave the spinal cord with the thoracic rather than with the sacral outflow of visceral nerves (*loc. cit.*, p. 27).

In the grey matter of the lower dorsal region and of the lumbar swelling of the cord there are aggregated a number of centres having to do with important functions, which may be variously interfered with by disease. These centres are those which regulate—(7) the evacuation of the rectum; (8) the evacuation of the bladder; (9) erection and ejaculatio seminis; and (10) the contractions of the uterus.

Gaskell says (*loc. cit.*, p. 17):—"The observations of Eckhard and subsequent observers have proved without doubt that that stream of visceral fibres which passes out of the sacral region, and forms the nerves called by them nervi erigentes (to which I have given the name of pelvic splanchnic nerves) contains motor nerves for the longitudinal muscles of the rectum, bladder and uterus, dilator nerves for the bloodvessels of the erectile tissue of the penis, motor nerves for Houston's muscle, secretory nerves for the prostrate gland, inhibitory nerves for the circular muscles of the rectum (Fellner and v. Basch); but no observer has yet discovered that it contains a single motor nerve for any bloodvessel." These nervi erigentes arise especially from the second and third sacral nerves.

In each case, the spinal centre constitutes an independent reflex centre, provided with its afferent and efferent nerves, but in each case, also, there must be anatomical connections between the spinal centre and others in the cerebral hemispheres. There must, therefore, be double sets of internuncial fibres for each centre traversing the whole length of the spinal cord and medulla; partly for the transference of afferent impressions from each centre to the brain, and partly for the conduction of efferent impressions in the reverse direction.

In the case of the uterine centre these cerebral connections are of comparatively slight importance; since, with a complete transverse lesion in the cervical or even in the upper dorsal region, the process of parturition may still be successfully accomplished. So long as the spinal mechanism is complete and perfect, parturition may take place without the need of cerebral co-operation. Our subsequent remarks will, therefore, refer principally to the other three lumbar centres.

Complete transverse lesions occurring in any part of the dorsal or cervical regions will, of course, cut off all the above-mentioned lumbar spinal centres from connection with, and therefore from any voluntary control exercised by, the cerebral hemispheres. But various limited focal lesions in particular transverse areas of the cord (though such areas cannot at present be definitely specified) may produce similar results, so far as the cerebral control of any one or two of the lumbar centres is concerned. According as the severance of these lumbar spinal centres from cerebral correlation and control is complete or partial, one or other of the following results would be produced :—

Name of Centre.	Complete Severance from Cerebrum.	Incomplete Severance from Cerebrum.	
	Afferent and Efferent Internuncial Fibres.	Afferent Internuncial Fibres Only.	Efferent Internuncial Fibres Only.
Rectal centre	Unconsciousness of need, and inability to prevent evacuation. *Result.* — Constipation, with incontinence of fæces after an aperient.	Unconsciousness of need and therefore no attempt to restrain evacuation.	Consciousness of need to evacuate, with no ability to restrain the act.
Vesical centre	Unconsciousness of need, and inability to prevent micturition. *Result.*—Reflex evacuation in gushes at intervals.	Unconsciousness of need and therefore no attempt to restrain micturition.	Consciousness of need, but inability to restrain micturition.
Sexual centre	Diminution or absence of sexual desire. Erections and emissions, if they occur, wholly dependent upon the spinal reflex mechanism.	With simple destruction of fibres, nearly same results as set down in previous column; but with *irritation* of afferent fibres there might be great increase of desire (satyriasis or nymphomania).	Feelings of desire, but no erection in response. Erection and emissions, if present, purely through spinal reflex. But with *irritation* of efferent fibres there may be persistent erections, mostly without desire.

The rectal and the vesical spinal centres are each composed of two parts with their separate afferent and efferent nerves —one in relation with a sphincter muscle, and the other in relation with detrusor or expulsive muscles in functional opposition with the former. These several nerve-fibres, both afferent and efferent, are largely contained in the sacral nerves—that is, many of them both reach and leave the lumbar swelling as constituents of these nerve-trunks. Destruction or irritation of either of these sets of fibres, or of one of the centres, will necessarily interfere to some extent with the working of this particular centre, so that its functions may be interfered with in several different ways. There may be various degrees of irritability of the bladder or rectum, or various degrees of paralysis of these organs.

In cases of paralysis of the bladder, especially when owing to lesions implicating its spinal centre, the urine soon becomes fœtid and alkaline, and inflammation (alone or with ulceration) is most apt to be set up in its mucous membrane.

The details as to the modes of disturbance of the genital function where disease implicates its lumbar centre, or the afferent and efferent nerves in connection therewith, are both less known and of less clinical importance than where it involves the internuncial fibres between this centre and the cerebrum. Again, should the lumbar portion of the cord become affected in a pregnant woman so as to involve the uterine centre, unless the contents of the womb were thrown off during some initial period of irritation, this organ would be quite incapable of expelling the fœtus and its accessories.

(*b*). *Evidence as to the Longitudinal Situation of the Lesion derivable from implication of Particular Sensory or Motor Nerves.*

The more precise indications concerning the longitudinal level at which the spinal cord is implicated are, as already stated, derivable from the level at which alterations in sensibility or in motility (either voluntary or reflex) are to be detected. The more closely the lesion approaches to what is called a 'total transverse lesion,' the more distinctly will signs of this order reveal themselves. It is important, too, to recollect that the fibres of different sensory roots are, to some extent, dispersed through cutaneous surfaces overlying the muscles supplied by the corresponding motor roots.

In regard to *sensibility*, the upper limit at which the trunk is affected is often sharply defined by the presence of a feeling of constriction, of pain, or of numbness ('girdle sensation') encircling the body. This sensation is generally supposed to be due to irritation of the roots of the nerves as they traverse the posterior columns (or perhaps, just outside them) at the upper level of the lesion. Such a symptom may, of course, be absent, but in many cases of paraplegia it is well-marked.

Efforts were also formerly made to define the muscles that were paralysed, in any given case, with a view to determine the upper limit of the lesion in the cord. A reference to the nerves by which such muscles are innervated ought to enable us, it was thought, to fix upon the particular segment of the cord from which such nerves proceed, and thus to determine with precision the upper level of the lesion in the motor regions of the cord.

These latter indications have been found, however, to be by no means so distinct as many might suppose, because the majority of limb and trunk muscles receive fibres from more than one motor root, as Preyer and Krause showed long ago. The view subsequently indicated, in fact, by E. Remak, that functionally related or synergic muscles are represented together in the anterior horns of the spinal cord has been confirmed and extended by Ferrier and Yeo (' Proceed. of Royal Soc.,' March, 1881, p. 12), by the experiments they have conducted with the view of determining the functional relations of the several motor roots in the brachial and crural plexuses.

They found that stimulation of individual roots of the brachial and crural plexuses resulted, not in mere unrelated contractions of various muscles, but in highly co-ordinated synergic contractions, leading to definite movements. But as the "muscles thrown into action by each root are innervated in most cases by several nerve-trunks," the result "of section of each motor root would therefore be paralysis of the corresponding combination, not necessarily, however, of the individual muscles involved . . . . whilst *weakened*, they might yet act in other combinations in so far as they were supplied by other roots."

The different combined movements which have been found to be dependent upon particular motor roots, by the authors of this valuable paper, are cited below.

THE BRACHIAL PLEXUS.

(Comprising the Roots of the Fourth to the Eighth Cervical Nerves, and the First Dorsal Nerve.)

Fourth Cervical.—The shoulder and upper arm are raised upwards and backwards, the forearm is flexed and supinated (and the wrist extended).

The action here is in other respects similar to that of the fifth cervical, except in the raising the arm upwards and backwards. The muscles observed in action were the deltoid, the rhomboid, the supra- and infra-

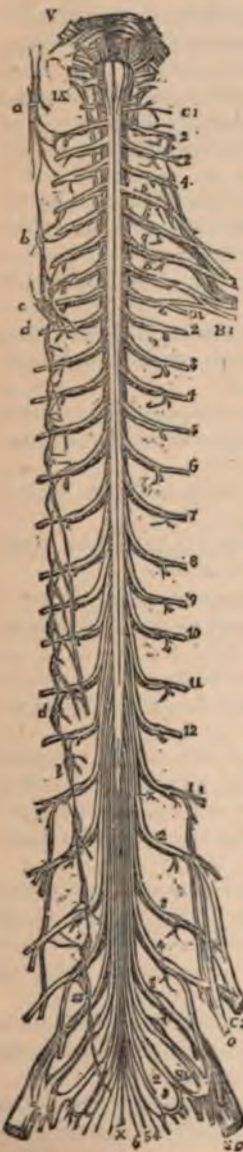

FIG. 129. DIAGRAMMATIC OUTLINE OF THE ROOTS AND FIRST PART OF THE SPINAL NERVES, TOGETHER WITH THE SYMPATHETIC TRUNK OF ONE SIDE [after Allen Thompson].

Front view. In the upper part of the figure the pons Varolii and Medulla oblongata are represented, and from V to IX the roots of the several Cranial Nerves from the Trifacial to the Hypoglossal are indicated.

On the left side Ci, is placed opposite the first cervical or suboccipital nerve; and the numbers 2 to 8, following below, indicate the corresponding Cervical Nerves.

Br, indicates the Brachial Plexus.

Di, is placed opposite the intercostal part of the first Dorsal Nerve; and the numbers 2 to 12 following, mark the corresponding Dorsal Nerves.

Li, the first Lumbar Nerve, and the numbers 2 to 5 following, the remaining Lumbar Nerves. Cr, the Anterior Crural, and *o*, the Obturator Nerve.

Si, the first Sacral Nerve, and the following numbers 2 to 5 the remaining Sacral Nerves. 6, the Coccygeal Nerve. Sc, Great Sciatic Nerve.

X, the filum terminale of the Cord.

On the right side of the figure the following letters indicate parts of the Sympathethic Nerve, viz.,

a, the Superior Cervical Ganglion communicating with the upper cervical spinal nerves and continued below into the great sympathetic cord.

b, the Middle Cervical Ganglion.

c, *d*, the Lower Cervical Ganglion united with the first dorsal.

d', the eleventh dorsal ganglion; from the fifth to the ninth dorsal ganglion the origins of the Great Splanchnic Nerve are shown.

l, the lowest dorsal or Upper Lumbar Ganglion.

ss, the Upper Sacral Ganglion.

In the whole extent of the Sympathetic Cord, the twigs of union with the Spinal Nerves are shown.

spinatus muscles, the flexors of the forearm and extensors of the wrist, though occasionally the last were not observed.

During stimulation of this root, respiration ceases from spasm of the diaphragm.

The action described, implies stimulation of the fibres conveyed by the rhomboid, supra-scapular, circumflex, musculo-cutaneous, and musculo-spiral nerves, and also of the phrenic.

Fifth Cervical.—The upper arm is raised upwards and inwards, the forearm flexed and supinated, the wrist and basal phalanges extended. The fingers assume a claw-like position with their distal phalanges bent.

The result of this action is to bring the hand up to the mouth. Among the muscles in action, there were specially noted the deltoid (its clavicular portion more particularly), the serratus magnus, and the flexors of the forearm—viz., the biceps, brachialis anticus, and supinator longus. The extensors of the wrist and basal phalanges were also in action.

The action of these various muscles implies stimulation of nerve fibres conveyed by the circumflex, musculo-cutaneous, musculo-spiral, and apparently also the median nerves (to long flexors of the fingers).

Sixth Cervical.—The upper arm is adducted and retracted, the forearm pronated and extended, the wrist flexed, and the palm of the hand brought against the pubes.

This movement generally occurs quickly, and the palm of the hand is brought smartly backwards towards the middle line.

It seems to be the action which, if the hands were the fixed point, would raise the body upon a trapeze or branch.

The contraction of the pectoralis, the latissimus dorsi, triceps, and flexors of carpus were noted. The pronators were also evidently in action, though not visible.

The action of these muscles implies stimulation of the external anterior thoracic, of the long subscapular, and of branches of the musculo-spiral and median nerves.

Seventh Cervical.—The upper arm is adducted, rotated inwards and retracted, and the forearm extended so as to bring the dorsum of the hand against the rump, the wrist and fingers flexed (at their second phalanges), so as to bring the tips of the fingers towards the radial side and against the rump.

The action here described is the scalptor ani movement, and involves the co-operation of numerous muscles. The teres major, lattissimus dorsi, and subscapularis appeared to be in action. The pectoralis major was noted in one instance also. The triceps was also observed to be contracting, as well as the long flexors of the fingers.

The action of these muscles would indicate stimulation of nerve fibres contained in the subscapularis, musculo-spiral, and median nerves—possibly also of the external anterior thoracic going to the pectoralis major.

Eighth Cervical.—A complex action comprising firm closure of the fist (intrinsic muscles and long flexors of fingers and thumb), pronation and flexion of the wrist (to the ulnar side), extension of the forearm with retraction of the upper arm (long head of the triceps in full action). The extensor muscles on the back of the forearm generally, were also, in two instances especially, observed to be rigid.

The action here described may be exactly imitated by pulling some object hanging in front downwards and towards the hip, or by drawing a scimitar from heel to point through some object lying in front.

The muscles involved imply stimulation of fibres conveyed in the ulnar, median, and musculo-spiral nerves.

The pectoralis major seems also to co-operate in this movement in man, but it was not specially observed by Ferrier and Yeo in their experiments, though the shoulder was noted to be depressed. This also would imply stimulation of the internal anterior thoracic, which anatomically is related to the cord formed by the eighth cervical and the first dorsal.

First Dorsal.—Adduction of the thumb, and flexion of the fingers at the metacarpo-phalangeal joints. The distal phalanges are slightly extended, and the fingers spread. The transverse diameter of the hand is diminished, and the dorsal aspect rendered more convex.

The action is that of the intrinsic muscles of the hand. Along with the action of the hand muscles there is also contraction of the muscles on the same side of the neck, causing the head to be drawn towards the shoulder.

THE LUMBO-SACRAL OR CRURAL PLEXUS.

(Comprising the Roots of the Third, Fourth, and Fifth Lumbar Nerves, and the First and Second Sacral Nerves, in Man.)

First Lumbar.—Stimulation of the equivalent of this nerve in man (viz., the second lumbar in the monkey) and of the last dorsal, caused contraction of some muscles in the flank and hypogastric region.

Second Lumbar.—Stimulation of the equivalent of this nerve in man (viz., the third lumbar in the monkey) caused contraction of the muscles in the flank, but no action in the leg. The cremaster muscle was not observed, though its contraction might also be expected.

Third Lumbar.—Flexion of the thigh on the pelvis and extension of the leg.—This brings the leg in the line straight forwards.

In addition to the ilio-psoas (evidently in action, though not visible), the sartorious, adductors, and extensor cruris were observed to contract. No action was observable in the muscles of the leg or foot.

The nerves involved are conveyed by the anterior crural and obturator trunks.

Fourth Lumbar.—Extension of the thigh, extension of the leg and pointing of great toe.—The combined result is straightening of the whole limb directly backwards, and seems to be the movement which immediately precedes the lifting of the foot to take another step forward in the act of walking.

The muscles observed in action were the gluteal, the adductors, the extensor cruris, and the peroneous longus. This latter explains the pointing of the great toe by depression of the base of the first metatarsal bone; and at the same time the raising of the outer edge of the foot. The sural muscles did not seem to contract.

These actions apparently involve stimulation of fibres conveyed by the superior gluteal, anterior crural, and obturator nerves, as well as by the musculo-cutaneous branch of the external popliteal.

Fifth Lumbar.—Rotation outwards of thigh (which assumes a position midway between extension and flexion), flexion of the leg with inward rotation, so that the foot points inwards, plantar flexion of the foot with flexion of the hallux and toes at their distal phalanges. The outer edge of the foot is somewhat raised.—This complex action involves the co-operation of many muscles difficult to analyse. We noted action of muscles in the gluteal region, the hamstrings, sural muscles, long flexors, the tibialis anticus and posticus, the peroneal muscles and also the extensors of the toes.

The action of these muscles involves stimulation of fibres from the trunk, and from the external and internal divisions of the great sciatic nerve.

First Sacral.—Flexion of the leg (hamstrings), plantar flexion of the foot (sural muscles), adduction of the hallux and flexion of the proximal phalanges (as in second sacral), with the addition of flexion of the hallux at the distal phalanx (long flexor).—The thigh is slightly rotated outwards, so that the plantar aspect looks towards the middle line.

The tibial and peroneal muscles and long flexors of the toes do not act.

The nerves in action are branches of the great sciatic and its internal popliteal divisions.

Second Sacral.—Adduction and flexion of hallux (basal phalanx), flexion of the proximal phalanges of the toes with slight separation and extension of the distal phalanges.—The action here is identical with that of the first dorsal in the brachial plexus, and is due to the intrinsic muscles of the foot.

In reference to these results Ferrier and Yeo write as follows:—"It will be seen that the movements which result from stimulation of the individual roots of the brachial and crural plexuses are not mere contractions, more or less strong, of various muscles (though many muscles are excited to contraction by more than one root, as previous experimenters have found), but a highly co-ordinated functional synergy in each case, as Remak has supposed. . . . The muscles thrown into action by each root are innervated in most cases by several nerve trunks, whence it would appear that the plexiform junctions of the various roots are for the purpose of distributing the requisite motor fibres in different trunks to the various muscles engaged in each functional combination. . . . The result of section of each motor root would, therefore, be paralysis of the corresponding combination, not necessarily, however, of the individual muscles involved. For, as many of these are innervated from more than one root, the degree of paralysis of the muscles would depend on the degree of motor innervation by the root divided; and, therefore, while weakened, they might yet act in other combinations in so far as they were supplied by other roots."

(c).—*Evidence as to the Longitudinal Situation of the Lesion derivable from the disappearance of one or more Spinal Reflexes.*

The reflexes of purely spinal mechanism which are of importance (by their presence, absence, or variation) as indications of disease of the spinal cord in different longitudinal regions have been divided into (1) the superficial or *skin* reflexes, and (2) the deep or so-called '*tendon* reflexes.'

(1). **Skin Reflexes.**—The most important of these are tabulated below. The designation of the parts of the cord upon which they severally depend is based upon a very useful table given by Gowers ('Diagnosis of Diseases of the Spinal Cord').

Name of Reflex.	Mode of Excitation.	Nature of Result.	Level of Cord upon which Reflex depends.
Plantar reflex.	Tickling sole of foot.	Movements of toes; of these and foot; or of toes, foot and leg.	1st, 2nd, and third sacral nerves (lower part of lumbar enlargement).
Gluteal reflex.	Irritation of skin of buttock.	Contraction of glutæi.	4th and 5th lumbar nerves.
Cremasteric reflex.	Irritation of skin of upper and inner part of thigh.	Drawing up of testicle.	1st and 2nd lumbar nerves.
Abdominal reflex.	Irritation of skin of abdomen along edge of ribs, and above Poupart's ligament.	Contraction of upper or of lower part of abdominal muscles.	8th to 12th dorsal nerves.
Epigastric reflex.	Stroking side of chest over 6th and 5th intercostal spaces.	A dimpling of corresponding side of epigastric region (contraction of highest fibres of rectus abdominus).	4th to 6th or 7th dorsal nerves.
Scapular reflex.	Irritation of skin in interscapular region.	Contraction of posterior axillary fold (teres), or of several of scapular muscles.	6th or 7th cervical to 2nd or 3rd dorsal nerves.

These skin-reflexes vary much in different individuals, as regards the facility with which they may be obtained. They are often more marked in children and in women than in men; though when the latter are of an irritable or nervous temperament, some or all of the skin-reflexes may in them be well-marked even in conditions of health.

In cases where extensive transverse lesions exist, situated higher in the cord than the nerves upon which any of these reflexes depend, such reflexes are commonly supposed to be exaggerated in intensity. This, however, is far from being always the case.

The fact that this or that reflex exists, shows not only that the afferent and efferent nerves, but that the track through the spinal cord at the corresponding level is practically undamaged. It is, however, not necessarily true that absence of either of the reflexes is an indication of disease at the corresponding level in the spinal cord. It may be so; but it may also be that the disappearance of the reflex is dependent upon disease in some part of the afferent or of the efferent nerves, leaving the cord itself intact. Or, it may also happen that the particular reflex is simply not to be obtained in the individual under examination. Again, with a total transverse lesion in the lower cervical region, nearly all reflexes dependent upon lower portions of the cord are abolished rather than exaggerated (see 7. *Softening of Spinal Cord*).

Further, it must be borne in mind that in certain cases of hemiplegia (even where hemianæsthesia does not coexist) these skin-reflexes are often notably diminished or even abolished on the paralysed side of the body; though the reverse condition of things will probably obtain in regard to the deep or 'tendon reflexes' next to be considered. It will probably be found, hereafter, that this repressing effect upon the skin-reflexes is associated with the existence of lesions in special parts of the brain, and not with lesions in other localities, though such several sites cannot at present be specified.

(2). **'Tendon reflexes.'** Much discussion has taken place as to whether these are 'reflexes' at all, in the proper sense of the term. Into this question we do not propose to enter here (see p. 222). The phenomena themselves, to which alone reference will be made, are chiefly two in number, namely, (*a*) ankle clonus; and that variously known (*b*) as the knee phenomenon, patellar tendon reflex, knee reflex, or knee-jerk.

There is a distinct difference in regard to these two phenomena. The 'knee-jerk' occurs in health, so that it is its absence which is of principal significance in certain diseases. 'Ankle-clonus,' on the contrary, is a phenomenon not to be obtained in a state of health, though it may occur, as Angel Money more especially has ascertained, in typhoid and other febrile diseases ('Lancet,' 1885, p. 842), altogether apart from definite disease of the brain or spinal cord. Its presence has therefore been erroneously thought by some to be a positive sign of disease of the spinal cord—a view which requires serious limitations, and limitations of such a kind as to deprive the manifestation of ankle-clonus of much of its diagnostic significance.

Ankle-clonus may exist, for instance, after one-sided fits dependent upon disease of the cerebral cortex ; and, again, it may exist to a well-marked extent where the antero-lateral columns of the cord are pressed upon at a certain level, even though (as in the condition above referred to) no lateral sclerosis of the cord has been developed. Again, it seems undoubtedly to occur in some cases of hysteria ; that is, in cases where functional perversion rather than structural defect exists.

Name of Reflex.	Mode of Excitation.	Nature of Result.	Level of Cord upon which Reflex depends.
Knee-jerk.	By striking patellar tendon with edge of hand or with percussion hammer whilst leg hangs loosely over fellow, or over forearm of operator. Also by striking quadriceps tendon, above patella.	A single upward jerk of the leg and foot, slight or distinct.	2nd and 3rd lumbar nerves.
Ankle-clonus	With knee extended or very slightly flexed, by pressing quickly and firmly against anterior part of sole of foot (so as to stretch calf-muscles) and then keeping up the pressure.	A series of clonic contractions at the ankle-joint, continuing as long as the pressure is maintained, and instantly ceasing when it is relaxed. If the condition is very highly marked it may spread to the whole limb, or even to that of the opposite side.	1st to 3rd sacral nerves (lower part of lumbar enlargement).

Both these physical signs have of late years attracted much attention. Ankle-clonus was originally described by Brown-Séquard in 1858; it was more particularly defined in the human subject by MM. Charcot and Vulpian in 1866 ; and its diagnostic importance has since been repeatedly enforced by these observers. In 1874 the mechanism of the knee-jerk, and the fact of its absence in certain spinal diseases, especially locomotor ataxy, began almost simultaneously to engage the independent attention of Erb and of Westphal, and subsequently of many other observers.

The integrity of those *reflex* actions which can be elicited either in health or in disease, depends, of course, upon the integrity of the entire nervous arcs concerned (that is, upon the integrity of ingoing fibres, of centres, and of outgoing fibres). Thus, though the impairment of a reflex *may not necessarily* be due to central causes, its unimpaired presence, on the other hand, clearly shows that the grey matter and other regions of the cord which must be traversed by its stimuli are not impassable; while its exaltation will indicate the probable existence of some central change, by which the grey matter in question is rendered more excitable, or else by which it is cut off from cerebral inhibitory influences.

(*d*).—*Relations of the Vertebræ to different Nerve Roots and different levels of the Spinal Cord.*

Seeing that the cord is liable to be implicated secondarily in cases where primarily there is disease or injury of the vertebræ it is needful to know the exact relations existing between the different vertebræ and the different nerve roots.

Only the highest cervical nerves arise from the cord opposite the place at which they leave the spinal canal. As we descend, the distance between these two points gradually increases, and it attains its maximum when we come to the nerves of the cauda equina. What nerve origins correspond, therefore, to a given vertebral level can only be decided by careful anatomical investigation.

A further complicacy is introduced into this question seeing that the vertebral spines, which we are compelled to deal with as localising guides, have different relations in different parts of the spinal column to the bodies of their respective vertebræ.

Gowers, who, together with Horsley, has paid special attention to these questions, has arrived at the following conclusions.

Relations of Spines to Bodies of Vertebræ.—The tips of the cervical spines correspond nearly to the lower borders of the corresponding vertebræ.

Each of the upper three dorsal spines corresponds nearly to the upper border of the body of the vertebra below. From the 4th to the 8th dorsal, each spine corresponds to the middle of the body of the vertebra below. The 9th, 10th, and 11th spines slope less, so that their tips again correspond with the upper borders of the next vertebræ.

The rest of the spines are opposite the bodies of their own vertebræ.

Relations of Vertebral Spines to Origins of Nerve Roots.—The first three cervical spines are opposite the origins of the 3rd, 4th, and 5th cervical nerves.

The 6th and 7th pairs arise opposite the intervals between the 4th and 5th, and the 5th and 6th, cervical spines respectively.

The 6th cervical spine corresponds with the origin of the 8th cervical nerve.

The 7th cervical spine corresponds with the origin of the 1st dorsal nerve.

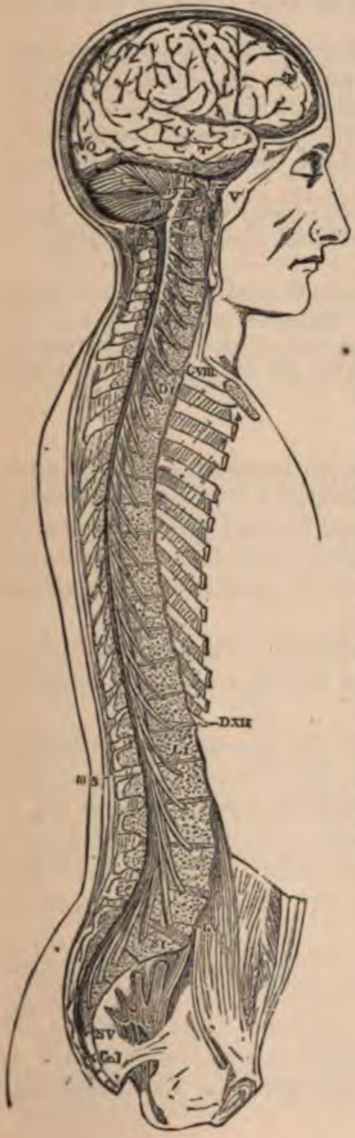

FIG. 130.—VIEW OF THE CEREBRO-SPINAL AXIS [after Bourgery].

The right half of the cranium and trunk of the body has been removed by a vertical section; the membranes of the right side of the Brain and Spinal Cord have been cleared away, and the roots and first part of the fifth and ninth cranial nerves and of all the spinal nerves of the right side, have been dissected out and laid separately on the wall of the skull and on the several vertebræ opposite to the place of their natural exit from the cranio-spinal cavity.

F, T, O, Cerebrum. C, Cerebellum. P, Pons Varolii. *m,o*, Medulla oblongata. *ms*, *ms*, Point to the upper and lower extremities of the spinal marrow. *ce*, On the last lumbar vertebral spine marks the cauda equina.

V, The three principal branches of the nervus trigeminus. Ci, The sub-occipital or first cervical nerve. Cviii, The eighth or lowest cervical nerve. Di, The first dorsal nerve. Dxii, The last dorsal nerve. Li, The first lumbar nerve. Lv, The last lumbar. Si, The first sacral nerve. Sv, The fifth sacral nerve. Co i, The coccygeal nerve. *s*, The left sacral plexus.

The first four dorsal spines vary. The 1st spine corresponds to the interval between the 2nd and 3rd pairs, or to the origin of the 3rd pair. The 2nd spine is between the 3rd and the 4th pairs, or opposite the 4th pair. The 3rd spine is opposite the 5th, or the interval between the 5th and 6th pairs. The 4th spine is opposite the lower part of the origin of the 6th pair, or even below it.

The 5th dorsal spine always corresponds to the origin of the 7th pair; the 6th spine to the 8th pair; the 7th to the 9th pair; the 8th to the upper part of the 10th pair; the 9th to the 11th pair; and the 10th to the 12th pair.

The 1st lumbar nerve arises opposite the 11th dorsal spine; the 2nd lumbar opposite the interval between the 11th and 12th spines; the 3rd and 4th lumbar nerves opposite the 12th dorsal spine.

The 5th lumbar and 1st sacral nerves arise opposite the interval between the 12th dorsal and the 1st lumbar spines; whilst the remaining sacral nerves arise nearly opposite the 1st lumbar spine.

Thus the cervical enlargement of the cord, which ends at the origin of the first dorsal nerves, corresponds nearly to the bodies and spines of the lower five cervical vertebræ; and the lumbar enlargement, which commences at the 12th dorsal nerves, corresponds with the 10th, 11th, and 12th dorsal and 1st lumbar spine.

SUMMARY OF DATA REFERABLE TO THE EXISTENCE OF DISEASE IN DIFFERENT LONGITUDINAL SEGMENTS OF THE SPINAL CORD.

For practical purposes it will be well to group together the various indications as to longitudinal localisation to which we have referred—classifying them as they are related to one or other of four imaginary segments of the spinal cord.

(*a*).—**Cervical Region of the Spinal Cord.**—*This corresponds externally to the space between the Occiput and the upper border of the 7th Cervical Spine (8th Cervical Nerve).*

The 1st, 2nd, and 3rd cervical spinous processes are respectively opposite the origins of the 3rd, 4th, and 5th cervical nerves. The phrenic nerve (motor nerve of the diaphragm) arises from the 4th, or from the 3rd and the 4th cervical nerves. Opposite the 3rd cervical spine (level of 5th cervical nerve) the cervical swelling of the cord begins; whilst it ends opposite the 7th cervical spine (level of 1st dorsal nerve).

Disease of this region may involve interference with respiration, and possibly weakness of voice; interference with the heart's action—pulse very frequent, or the reverse; flushing or pallor of the head and neck; continued priapism (with crushing lesions); augmentation of temperature in the body generally (hyperpyrexia); and marked contraction or dilatation of the pupil.

The nerves for the shoulder, arm, and hand muscles are derived from spinal nerves between the 6th cervical and 1st dorsal inclusive; those supplying the ulnar side of the hand and forearm arising from the lower level, that is, from the upper part of the next region.

(*b*).—**Upper half of the Dorsal Region of the Spinal Cord**.—*This corresponds externally to the space between the 7th cervical spine (1st dorsal nerve) and the 4th dorsal spine (6th dorsal nerve).*

The results of disease here are apt to be these:—The 'scapular reflex,' may be abolished, calling into activity, as it does, the last two or three cervical and the first two or three dorsal nerves; the intercostal muscles are paralysed at different levels; a 'girdle sensation' is felt at different levels; there may be prominence of certain vertebral spines, and possibly tenderness on pressure or on tapping over them; the 'epigastric reflex' may be abolished, depending as it does upon the spinal cord at the level of the 4th to the 6th or 7th pairs of dorsal nerves; and priapism (with crushing lesions) may occasionally be met with.

(*c*).—**Lower half of the Dorsal Region of the Spinal Cord.**—*This corresponds externally to the space between the upper border of the 5th dorsal spine (7th dorsal nerve) and the lower border of the 10th dorsal spine (space below 12th dorsal nerve).*

Disease here may give rise to the following symptoms:—The 'abdominal reflex' may be abolished, depending as it does upon the integrity of the cord between the levels of the 8th dorsal and the 1st lumbar nerves. Paralysis of lower intercostal muscles or of abdominal muscles may possibly occur, in addition to paralysis of the lower extremities. A 'girdle sensation' may be felt at different levels (the umbilicus corresponding with the 10th dorsal nerve, and the 'ensiform area' with the 6th and 7th dorsal nerves). There may be prominence of certain of the lower dorsal spines, with possible tenderness.

(*d*).—**Lumbar Region of the Spinal Cord.**—*This corresponds externally to the space between the lower border of the 10th dorsal spine (just below 12th dorsal nerve), and the upper border of the 2nd lumbar vertebra.*

Here the symptoms are:—Paralysis, not implicating the abdominal muscles, but limited to muscles pertaining to the lower extremities. No 'girdle sensations' around the trunk. Three superficial reflexes may be abolished, namely, the 'cremasteric,' which depends upon the integrity of the cord in the upper lumbar region, also the 'gluteal' and the 'plantar,' both of which seem to be dependent upon the integrity of the lower part of the lumbar region of the cord. A deep reflex may also be abolished, namely, the so-called 'knee-jerk,' which is dependent upon the upper lumbar region of the cord. 'Ankle-clonus' may be met with when disease affects the upper or mid-lumbar regions of the cord, but not where the lower lumbar region is implicated. Loss of sensibility about the perineum and anus (if not due to disease of nerve-trunks), is indicative of disease of the posterior columns in the lower lumbar region. Absolute paralysis of the bladder and rectum may be present, with tendency to inflammation and ulceration of the former organ.

The clinical data above given include the majority of the facts upon which the regional diagnosis of diseases of the spinal cord must in all cases be based. In them, also, will be found the explanations, so far as they can be given in moderate compass, of the majority of the *symptoms* met with in different diseases of the spinal cord.

PATHOLOGICAL DIAGNOSIS.

GENERAL CONSIDERATIONS CONCERNING THE SEVERAL CONDITIONS THAT CAUSE PARALYSES OF SPINAL ORIGIN.

The causes of Spinal Paralyses are primarily divisible into two great classes.

The spinal cord may be compressed or invaded by morbid conditions operating upon it from without (extrinsic causes), or it may be the seat of pathological conditions starting within its own structure (intrinsic causes).

The following causes of spinal paralyses may be ranged, therefore, under these two categories respectively :—

CAUSES OF SPINAL PARALYSES.

A. Extrinsic.

(Extra-Medullary).

1. Traumatisms.
2. Scrofulous caries of the Vertebræ.
3. Cancer and other affections of the Vertebræ.
4. Tumours of the Spinal Meninges.
5. Chronic inflammation of the Meninges (pachymeningitis).
6. Hæmorrhage into or upon the Meninges.

B. Intrinsic.

(Intra-Medullary).

7. Hæmorrhage.
8. Embolism.
9. Thrombosis.
10. Myelitis.
11. Primary scleroses.
12. Tumours in the Spinal Cord.
13. Atrophy with Degeneration of Ganglion cells.

A. EXTRINSIC CAUSES.

1. TRAUMATISMS.

Stabs or bullet wounds are particularly apt to involve limited regions and parts of the spinal cord.

Fracture with dislocation of some of the vertebræ (as results of severe falls or other mechanical violence) occasionally causes paraplegia, owing to pressure upon or actual crushing of the spinal cord. This is most apt to occur in the cervical region; to a less extent, the dorsal and lumbar regions are, however, liable to similar accidents.

2. SCROFULOUS CARIES OF THE VERTEBRÆ.

This may exist in either region, and may or may not be associated with angular curvature in the corresponding portion of the spine; such association is however most common in the dorsal region. The paraplegia, or other result of interference with the functions of the cord, at times met with in this malady (Pott's disease) is not due to its compression by diseased bone, but rather to irritation and subsequent compression of the cord by inflammatory thickenings of the dura mater or actual scrofulous growths therein (4, *b*).

3. CANCER AND OTHER AFFECTIONS OF THE VERTEBRÆ.

Cancer may occur either as a primary or as a secondary affection. Such a new growth may infiltrate the dura mater or not; and, as it grows, it may at first irritate and subsequently compress the spinal cord.

Other diseases of the spine are rare as causes of disease of the spinal cord; still, occasionally, enchondromatous growths, syphilitic hyperostoses, or rheumatoid arthritis affecting the vertebræ may give rise to some compression of the spinal cord. Very rarely, also, aneurysmal erosion of vertebræ may go so far as subsequently to permit of pressure upon the cord, and possibly also the aneurysm of the aorta may burst into the spinal canal.

4. NEW GROWTHS IN THE SPINAL MENINGES.

These may be of various kinds, and under this head we may also include such cancerous or sarcomatous growths as originate in the fibro-adipose tissue just outside the dura mater, as well as hydatid cysts likewise occasionally occurring in this situation.

(a). **Cancer.**—Cancer occurs in the spinal meninges most frequently as an extension from a previous cancerous growth in one of the adjacent vertebræ, rather than as a primary affection. The space within the spinal canal being so very limited, such a growth soon begins to press injuriously upon nerve roots and upon the spinal cord itself; and thus adds to the symptoms that may have been produced by the cancer of the vertebræ alone.

(b). **Scrofulous Growths.**—These masses are met with principally in cases of scrofulous disease of the spinal column (Pott's disease), and especially where angular curvature is produced, though they are not confined to these more severe forms of vertebral caries. Growths with a tendency to speedy caseation, are, in such cases, apt to extend from the vertebræ so as to infiltrate the dura mater, and then produce fungating excrescences on its inner surface. Such growths may oftentimes be classed, just as appropriately, under the head of 'spinal pachymeningitis.'

Small isolated scrofulous tumours, or typical tubercular growths, may also be met with (though more rarely than in the cerebrum) springing from the spinal pia mater, and more or less imbedding themselves in the substance of the spinal cord.

(c). **Syphilomata.**—Syphilitic growths are also decidedly less frequent in connection with the spinal than with the cerebral meninges. Small gummatous growths may, however, spring from the spinal dura mater or from the pia mater. At other times, instead of well-defined tumours there may be thickenings of the membranes in some part of their extent, and adhesions between one another, or of the pia to the surface of the cord, by means of opaque, yellowish-white gummatous material.

(d). **Sarcomata.**—Sarcomatous tumours of all kinds may be met with in connection with the spinal meninges, springing occasionally from the dura mater, but more commonly from the pia mater.

Instead of being distinctly circumscribed, such growths may exist in the form of diffuse infiltrations, invading the pia mater all round the cord for a variable extent. In one remarkable case I met with a growth of this kind involving the pia mater throughout the whole extent of the cord. The layer of new growth was rather thicker on the lateral and posterior aspects of the cord than elsewhere; here, in some places, it was as much as one third of an inch in thickness, and the cord beneath was notably compressed.

(e). **Myxomata.**—Myxomata are encountered occasionally in the form of small circumscribed tumours, springing mostly from the pia mater. I have seen one about the size of an almond, situated on and greatly compressing the posterior columns of the cord.

The other forms of new growth met with in connection with the meninges are either so small, or so rare, as to be scarcely worthy of mention here.

(f). **Hydatids.**—Hydatids are occasionally found in connection with the spinal meninges—especially where they also exist in other parts of the body. Hydatid cysts may be found within the dura mater, but they are more frequently encountered outside this membrane, in the cellulo-adipose tissue between it and the vertebral canal. They sometimes give rise to large swellings in this situation.

5. CHRONIC INFLAMMATION OF THE MENINGES (PACHYMENINGITIS).

In the case of the cerebral meninges, inflammation as a result of traumatic injuries is more common than as a phenomenon secondary to disease of the bone or of the scalp. The proportional frequency of such modes of causation is, however, somewhat reversed in the case of the spinal meninges; partly because the head is more liable than the spine to suffer from direct injuries, and partly because disease of the spine and adjacent parts occurs with considerable frequency in such a manner as to be capable of exciting a secondary inflammation of the spinal meninges.

Chronic inflammatory conditions of the spinal dura mater are the changes belonging to this class which, by far the most frequently, lead on to the production of paralyses of spinal origin.

In spinal pachymeningitis the dura mater itself is thickened and more vascular than natural, this being seen more especially on its outer surface. Such inflammation may be either limited to the region of two or three vertebræ, or it may affect more or less the whole length of the spinal membrane. The most common form of spinal pachymeningitis is that occurring in association with scrofulous disease of the vertebræ which not unfrequently acts as a cause of paralysis. This form of scrofulous inflammation or new growth in the spinal dura mater has already been referred to (p. 547).

Another more idiopathic, and also more chronic, form of pachymeningitis is that which has been observed by Charcot and others, as involving principally the inner layers of the dura mater in the cervical region. In this condition, which is described by the author above named as 'hypertrophic cervical pachymeningitis,' there seems to be a considerable hyperplasia of tissue elements in the inner layers of the dura mater, which are apt to develop into an overgrowth of almost cicatricial hardness, frequently made up of concentric laminæ. The dura mater in such cases often becomes adherent to the arachnoid and pia mater, these membranes becoming themselves also more or less thickened. In the latter cases more especially, not only are the spinal nerve roots greatly damaged, but the spinal cord is itself more or less compressed and softened, so that distinct paralytic symptoms, with muscular atrophy is apt to be produced. The thickening of the dura mater seems to begin anteriorly, and it is here, in the cervical region, that the membrane is naturally most closely connected by fibrous tissue to the posterior common ligament. I have also seen a cancerous thickening of the dura mater in this situation produce a 'cervical paraplegia' which proved fatal in the course of a few months.

6. HÆMORRHAGE INTO OR UPON THE SPINAL MENINGES.

Effusions of blood upon, between, or beneath the spinal meninges are altogether rare events, contrasting notably in this respect with the comparative frequency of parallel conditions on the side of the cerebral meninges.

Fluid blood, or blood clot, may exist in relation with the spinal meninges in three different situations.

The most frequent site of such hæmorrhages is (1) outside the dura mater, between it and the vertebral arches. Here large clots are sometimes found, wholly, or, more frequently, in part, surrounding the dura mater in the region in which the hæmorrhage has occurred. Where the effusion is large, the cord itself may be distinctly compressed; but even small effusions may produce some amount of compression of nerve-roots.

Such hæmorrhages are determined, perhaps, most frequently by falls or blows upon the spine.

Blood may also, but more rarely, be effused (2) inside the dura mater, between it and the arachnoid. This kind of hæmorrhage does not often produce definite spinal symptoms, since it occurs most frequently as a mere sequence of a similar hæmorrhage taking place into the cerebral meninges—the blood simply gravitating into the spinal canal.

In cases of spinal pachymeningitis, however, where the internal surface of the dura mater is much more vascular than natural, a bleeding may occur in this situation. Again, the opening of a thoracic or abdominal aneurysm may occasionally take place into the spinal canal, and thus produce a sudden and grave compression of the cord by blood poured out between these two membranes.

Much smaller extravasations are also occasionally met with (3) beneath the spinal pia mater, over areas perhaps small in extent longitudinally, but more or less completely embracing the cord in one or more regions. The cord or nerve-roots may, however, be decidedly compressed by such hæmorrhages, even when they are small in amount, owing to the narrowness of the space into which the effusion takes place.

In some of the cases of bleeding in this situation, the hæmorrhage seems to have been induced during violent straining efforts, such as may be put forth, for instance, during the lifting of heavy weights. Andral records a case of this kind, and I have also seen two or three.

The foregoing groups of causes of disease of the spinal cord, give rise to sets of symptoms having a generic resemblance, because in each case compression acts upon the cord, or upon the spinal roots and cord, from without, in one or other direction.

B.—INTRINSIC CAUSES.

7. HÆMORRHAGE.

Hæmorrhage occurs with extreme rarity in the substance of spinal cord. This is due, in the main, to the firmer texture of the cord as compared with the brain, and to the greater abundance of supporting connective tissue around its blood-vessels. When hæmorrhage, of idiopathic origin, does take place into the spinal cord, it almost invariably occurs in the softest portion of the organ, namely, its central core of grey matter—and in this region it may extend for some distance upwards and downwards.

As a result of falls or blows, also, hæmorrhage into the substance of the cord is a rare event; still, under these conditions, it occurs occasionally—mostly in association with laceration of the substance of the cord. Of this latter kind of lesion, resulting from a fall from a height of about 25 feet, the writer has recorded a remarkable instance ('Med.-

Chirurg. Trans.,' 1867) in which, although the cord was lacerated, there was no external wound and no fracture or dislocation of vertebræ.

8. EMBOLISM.

Embolism occurs with great rarity in the spinal cord, and is still more seldom recognized when it does occur. This is due to the fact of the small size of the arteries of the cord—the absence among them of any large trunk, like the middle cerebral, coming off more or less directly from one of the great vessels arising from the arch of the aorta. Emboli reach the brain much more rarely by way of the vertebrals than by way of the carotids; and the principal arteries of the spinal cord are either direct offsets from the vertebral (anterior spinal), or indirect branches from the same (posterior spinal). Apart from these vessels, the blood-supply of the cord comes from smaller twigs (still less likely to convey embolic particles), derived principally from the intercostal and lumbar arteries.

Embolism is, therefore, not to be considered as one of the causes of paralyses of spinal origin.

9. THROMBOSIS.

Thrombosis is capable of occurring in diseased spinal arteries, as well as in those of other parts of the body. The condition of the arterial supply of the spinal cord, indeed, to which Moxon has called particular attention (p. 498), would seem greatly to favour such a process. Subsequent observations may perhaps show that degenerative changes or endarteritis are particularly common in the spinal arteries, so that the occurrence of thrombosis in them would thereby be rendered all the more easy and likely to occur.

Thrombosis may also take place in the peculiarly tortuous network of veins which surrounds the spinal cord on all sides—perhaps even with more facility than in the veins of other parts—when general and other local conditions favour its occurrence. Ollivier called attention to the probably natural slowness of the blood-current through the spinal veins, and to the multiplicity of causes which, owing to their influence upon respiration and cardiac action, tend still further to retard it—such as violent emotions or efforts, and those diseases which greatly interfere with respiration, or with the force and regularity of the heart's action. He adds that he has often seen in elderly persons fibrinous clots filling the veins of the cord as well as those which accompany its nerve-roots.

Thus one of the common causes of ordinary degenerative or ischæmic softening as it occurs in the encephalon, is undoubtedly operative in the cord, and I must, therefore, express my strong dissent from the statement made by Charcot (' Localisation,' p. 41) to the effect that "softening secondary to arterial obstruction, whether it be due to thrombosis or to embolism, are almost unknown accidents in the spinal cord."

White softening of the spinal cord is very common; often implicating its whole transverse area for a variable extent. It differs in no respect in its naked-eye or microscopical appearances from the process as it is met with in the encephalon. It is altogether unreasonable to assume, in accordance with current nomenclature, that this condition is mostly a result of inflammation and therefore to be spoken of as a myelitis, when pathologists generally have agreed that the similar process in the cerebrum and cerebellum is mostly of degenerative origin.

10. MYELITIS.

The writer is far from denying that a primary inflammation may involve areas of the cord, and entail ' softening ' of its substance. He believes, however, that ' acute myelitis ' is far more likely to occur as a secondary process, in connection with pressure upon, and consequent irritation of, some part of the cord encroached upon by fractures and dislocations of the vertebræ, or when otherwise wounded; also as an occasional sequela either of scrofulous pachymeningitis, or of the direct pressure made upon the cord by some meningeal tumour, or of hæmorrhage into its substance. Yet he is far from believing that *all* the secondary softenings met with in the spinal cord are necessarily of inflammatory origin. Many of these, also, are due to degenerative rather than to inflammatory processes.

Processes of degenerative ' softening ' are mostly brought about quickly, and they would, in the main, correspond with what is commonly spoken of as ' acute myelitis.' As for ' chronic myelitis ' (in the commonly understood sense of chronic 'softening) the writer believes that no such disease should be any longer described. Many ' softenings ' are, in a certain sense, chronic, as, though they may be more or less abrupt in their onset, they tend to last long, rather than to kill quickly. Again, other maladies which the older physicians would have ascribed to ' chronic myelitis ' or ' chronic softening,' are now known to partake more of the nature of chronic indurations, and to have as their bases processes of sclerosis.

11. PRIMARY SCLEROSES.

Processes of sclerosis are extremely common in the spinal cord. In nature they are over-growths of the connective tissue of this organ, altogether similar to those occurring in other organs and tissues, under the name of 'fibroid substitutions' or 'non-inflammatory hyperplasias of connective tissue.' Yet here, again, certain pathologists would have us see results of inflammation, and they accordingly speak of such changes as examples of 'chronic myelitis.'

Sclerosis occurs under various forms, and constitutes the basis of several distinct diseases, which are, in all cases, gradual and more or less slow in their onset, as well as in their progress. It may occur (*a*) as a *diffuse* general overgrowth (after the manner of a cirrhosis in other organs) ; (*b*) in the form of *bands* limited to particular columns of the cord (especially the posterior and the lateral); or (*c*) in an *insular* manner, so as to form islets of sclerosis, scattered altogether irregularly through the cord at different levels, as in 'disseminated sclerosis.'

Tissue-changes more or less allied to these in their results or later stages, though they have a peculiar history and course of their own at the commencement, are met with in the **'secondary degenerations,'** already described as occurring in certain regions of the cord as a result either of some previous damage or injury to the organ itself, or as a sequence of brain-disease (pp. 197, 513).

These 'secondary degenerations' illustrate facts originally made known by Waller, but which were confirmed and extended by Phillipeaux and Vulpian, to the effect that when nerve-fibres are severed from their connections with ganglion-cells situated at one or other extremity, the white substance of Schwann gradually breaks up in the course of from seven to fourteen days, and undergoes a process of fatty degeneration, by which it is ultimately resolved into a multitude of mere molecules and fat particles. At the same time the neuroglia between the nerve-fibres begins to overgrow and thus leads on to the sclerosis which ultimately becomes established.

The white columns of the cord are, as is well known, composed of great aggregations of nerve-fibres bound together by neuroglia, and running, for the most part, parallel with one another, so that when one of these columns is cut across, or when the continuity of its fibres is completely interrupted by some severe lesion occurring in their midst, a process of 'secondary degeneration' manifests itself simultaneously in all the fibres thus damaged ; and the united result appears as a band-like tract of degeneration, running upwards or downwards in the particular column of the cord affected.

These tracts of degeneration have been already fully described. Of themselves, they are rarely causes of morbid symptoms; any symptoms with which they are associated are due rather to the antecedent conditions which cause the degenerations.

Occasionally, however, the process of sclerosis, constituting part of the pathological changes comprised in a tract of secondary degeneration, extends beyond the confines of such tract, so as to involve the contiguous grey matter of the anterior cornu. The result of this is, the production of a further pathological change, viz., atrophy in the muscles in relation with the damaged anterior cornu (p. 232).

Such hyperplasias of connective tissue in tracts of secondary degeneration are sometimes regarded as inflammatory in nature, so that the whole change has been spoken of by some writers as a 'chronic myelitis.' As previously indicated (p. 201) there is, in my opinion, little justification for such a view.

12. TUMOURS IN THE SPINAL CORD.

New growths in the substance of the spinal cord itself are not very common, nor, on account of the limitations of space within the spinal canal, do they ever attain a very large size. For this situation a growth equalling a hazel-nut in bulk may be esteemed large. In regard to the nature of the growth, this is a matter of almost purely pathological interest, since the clinical signs and symptoms which a growth in the spinal cord is capable of causing would not vary with its nature, but would be wholly dependent upon its situation and its rate and manner of increase.

Cancer occurs within the spinal cord almost solely as a secondary extension from a similar growth pre-existing in the dura mater or in the vertebræ, or possibly in more distant parts. In altogether exceptional cases it may occur primarily in the spinal cord.

Gliomata, sarcomata, and **myxomata** may also occasionally be met with, either in pure or in blended types.

Tubercular or **scrofulous** nodules are also apt to occur, either alone or in combination with a tubercular meningitis.

Syphilitic gummata may likewise be found in the substance of the cord, though their presence in this situation is not so frequent as it is in association with the spinal meninges.

13. ATROPHY WITH DEGENERATION OF GANGLION-CELLS.

Atrophy with degeneration of ganglion-cells is apt to occur as a secondary process, with extreme frequency, in any portions of the grey matter of the cord that happen to be more or less implicated by other contiguous pathological changes.

But in two or three distinct diseases the ganglion-cells of the anterior cornua, in different parts of the cord, are prone to be suddenly overtaken by an etiologically obscure and altogether inexplicable failure of nutrition, which speedily entails an atrophy of the particular cells affected. This, for instance, occurs as the anatomical basis of 'acute spinal paralysis,' both as it presents itself in children and (though more rarely) in adults; it is met with also in 'chronic spinal paralysis.' In these diseases whole groups of contiguous and functionally-related cells are affected simultaneously, and, as the atrophy progresses, there is generally evidence of a secondary overgrowth of the neuroglia surrounding such nerve-cells in the anterior cornua.

Of late some more evidence has been forthcoming to show that this process may be, at first, inflammatory in type, so that the terms 'cornual myelitis' or 'anterior polio-myelitis,' which have also been applied to these affections, are more in accordance with known facts than was formerly the case. Still, it may be said that inflammation does not usually limit itself to individual tissue-elements, and the slight overgrowth of the contiguous neuroglia may well be a secondary simple hyperplasic process. This latter process is, indeed, less evident where, as in 'progressive muscular atrophy,' the initial and mysterious atrophy of individual ganglion-cells occurs more slowly and more sparsely. In this affection we have cells, here and there, in particular groups, undergoing the atrophic process, leaving others around them, for a time, as healthy as ever. Yet, as the disease progresses, the ranks of the healthy cells become gradually thinned in an altogether irregular manner; and this atrophy of nerve-cells, as it occurs, speedily entails, for reasons that have been already set forth (p. 234), a corresponding atrophy of functionally-related muscular fibres.

PATHOLOGICAL DIAGNOSIS.

CLINICAL INDICATIONS FAVOURING THE EXISTENCE OF THIS OR THAT CAUSATIVE CONDITION.

The pathological diagnosis in any given case of spinal disease is dependent upon its apparent mode of causation; upon the mode (and, therefore, the rapidity) of the development of the disease; and upon the actual nature of the symptoms by which it is characterized. We have also to take into consideration the patient's previous and family history, as well as the present pathological condition, so far as this can be ascertained, of some of his principal tissues and organs.

The pathological diagnosis in the case of paralyses of spinal origin depends, therefore, upon the same kinds of facts as were found to be of maximum importance for arriving at a pathological diagnosis in the case of paralyses of encephalic origin. It will be found, however, as already indicated (p. 6), that the problems of regional and pathological diagnosis are rather more closely interwoven in the case of spinal diseases than they are in the majority of cases of encephalic disease, and this renders a different mode of treatment desirable in dealing with the present part of our subject (p. 559).

The first and most generally useful classificatory distinction available in the case of the pathological causes of spinal paralyses, is that based upon the rapidity of their operation or development. Some of the causes operate suddenly or abruptly; some cause acute diseases, such as develop in a few hours, or at most in the course of a few days; some cause sub-acute diseases, which develop within a few weeks; others cause chronic diseases, developing in the course of a few months; and others still very chronic diseases, in which the time needed for their establishment amounts to periods of six months or more.

These are, of course, rough arbitrary distinctions, and if, for practical purposes, we attempt to tabulate the different causes of spinal paralyses after such a fashion, such a classification must only be taken for what it is worth—viz., as a first rough grouping of spinal diseases, with the view of giving the student some notions as to the average comparative rapidity with which the several causes come into operation. It must clearly be understood, however, that exceptions to such a classification are to be met with, not unfrequently, and in the following table those pathological conditions which are more variable in the rapidity of their evolution and which would, therefore, be apt to range themselves sometimes under one and sometimes under another category, have this variability indicated by being placed between, rather than immediately under, either of the corresponding headings.

TABLE I.

Relative Rapidity of Action of the Several Conditions which Cause Paralyses of Spinal Origin.

<table>
<tr><th colspan="2"><i>Abrupt.</i>
(Minutes.)</th><th colspan="2"><i>Acute.</i>
(Hours or Days.)</th><th colspan="2"><i>Sub-acute.</i>
(Weeks.)</th><th colspan="2"><i>Chronic.</i>
(Months.)</th><th colspan="2"><i>Very Chronic.</i>
(Half-years.)</th></tr>
<tr><td colspan="2" rowspan="2">Traumatisms.
Meningeal Hæmorrhages.
Intra-medullary Hæmorrhages.
Ischæmia of Lumbar Swelling.</td><td colspan="2" rowspan="2">Thrombotic Softening.
Myelitis (Acute Cornual).</td><td colspan="2">Myelitis (Sub-acute Cornual).</td><td colspan="2">Cancer of Vertebræ, etc.</td><td colspan="2">Scleroses.</td></tr>
<tr><td colspan="3">Vertebral Caries (Pachymeningitis).</td><td colspan="3">Cervical Pachymeningitis.
Tumours of Spinal Meninges.
Tumours in Spinal Cord.
Atrophic Degeneration of Ganglion-cells.</td></tr>
</table>

It would serve no useful purpose to follow up here, by further analysis, the more detailed indications testifying to the existence of this or that pathological condition, as was done when treating of the different causes of paralyses of encephalic origin.

The subjects are, in the two cases, so different as to make a different treatment essential. The spinal cord is not like the encephalon, or, certainly, not to anything like the same extent, a congeries of extremely different parts, from the point of view of function. In many respects, too, our knowledge of spinal diseases is distinctly in advance of our knowledge of encephalic disease.

It is because of our commonly more complete power of regional diagnosis in diseases of the spinal cord, and because the regional diagnosis (especially as regards the longitudinal level of the cord involved) obtrudes itself at once upon our notice, that we have dealt with the problems involved in a regional diagnosis before considering those facts which are more immediately related to the pathological diagnosis.

Further, the extremely close interweaving of the problems concerning regional diagnosis with those related to the pathological diagnosis, makes it desirable not to pursue the latter in any detail alone—but rather to deal now with the two sets of problems conjointly, and to endeavour to indicate, therefore, the points to be considered in the differential diagnosis of the several Diseases of the Spinal Cord.

COMBINED REGIONAL AND PATHOLOGICAL DIAGNOSIS.

SYNTHESIS OF DATA BEARING UPON THE REGIONAL AND PATHOLOGICAL DIAGNOSIS, AS PRACTISED IN THE ACTUAL DIAGNOSIS OF THE SEVERAL DISEASES OF THE SPINAL CORD PRODUCTIVE OF PARALYSIS.

We must now gather up and combine the data previously set forth, in order to show how the different diseases of the spinal cord are to be distinguished from one another.

In any case of doubt, the student is advised first of all to consult Table I., in which the several pathological causes are ranged in the approximate order of their acuteness or chronicity. This may serve as a kind of preliminary index from which the first clue may be obtained, before resorting to the other differential tables now about to be given.

The first of these tables to which reference must be made (Table II.) is a general classified enumeration of the several spinal diseases with which we are concerned as producers of paralysis.

Only a few words of explanation are needed in order that the student may understand two of the classificatory terms employed. The term 'systemic lesion,' introduced by Vulpian, is now commonly adopted as a designation of those intra-medullary lesions which are strictly limited to one set of homologous parts in the spinal cord; that is, to one anatomical system of the cord, such as the posterior or lateral columns, or the anterior cornua. As a set off against this term, Bramwell, not inappropriately, designates the other intra-medullary organic causes of spinal paralyses 'indiscriminate lesions'—merely meaning to imply thereby, that they are not necessarily limited to any particular anatomical system or part of the spinal cord.

The five great groups into which the several spinal diseases or disorders productive of paralysis are here ranged, are not natural groups or genera from the point of view of diagnosis. They are rather generic groups from the point of view of etiology and pathology. This table is intended to serve, therefore, merely as an index of the affections with which we have to deal. The means by which they are to be discriminated from one another must now engage our attention.

For diagnostic purposes we may most usefully rearrange the several diseases as we did the several pathological causes themselves, into different groups according to their varying degrees of acuteness or chronicity. From this point of view, spinal diseases productive of paralysis, may be arranged in the different groups represented in Table III.

The several diseases are here broken up into natural groups based upon a practical distinction which suffices fairly well to distinguish them from one another.

It is the most useful primary ground of classification that can be adopted, since it should not be difficult to determine in any given case that comes before us, under which of these categories the patient's malady would fall. Of course, if the case has been under our own observation throughout, or under that of some competent professional observer, there would be no difficulty in this respect. But even in those very numerous cases in which the patient comes before us with his malady already more or less fully developed, and where we are thrown back for information as to its mode of onset upon what the patient himself or his friends can tell us, it is precisely this kind of information that we have the best chance of eliciting with some accuracy. Even the vaguest of patients can generally give us some information as to whether it took minutes, hours, days, weeks or months for his paralysis to become developed.

We must take the different groups in detail, and see by what characters the several diseases comprised under them may be diagnosed from one another. For a tentative diagnosis the student will do well to make use more especially of Table III., and Table IV., and then seek to render his diagnosis more certain by reference to the fuller details now to be given concerning the several diseases of the spinal cord.

TABLE II.

CLASSIFIED LIST OF SPINAL DISEASES ASSOCIATED WITH PARALYSIS.

Spinal Paralyses dependent upon recognizable Organic Causes.	Extrinsic Causes (Extra-Medullary Lesions)	A. *Traumatisms.*		Fractures and Dislocations of Vertebræ.	
				Punctured or Gun-Shot Wounds of Cord.	
				Concussion of the Spinal Cord.	
		B. *Slow Compressing Lesions.*	Vertebral Diseases.	Scrofulous Caries.	
				Cancer.	
				Enchondroma.	
				Rheumatoid Arthritis.	
				Syphilitic Hyperostosis.	
				Cervical Hypertrophic Pachymeningitis.	
				Tumours, etc., in Meninges.	
				Meningeal Hæmorrhage.	
	Intrinsic Causes (Intra-Medullary Lesions)	C. *System Lesions.*	Cornual	Acute Spinal Paralysis (Anterior Cornual Myelitis).	*α.* Of Children.
					β. Of Adults.
				Acute Ascending Paralysis.	
				Subacute and Chronic Spinal Paralysis.	
				Progressive Muscular Atrophy.	
				Pseudo-hypertrophic Paralysis ?	
			Columnar	Lateral Sclerosis (Spastic Paralysis).	
				Amyotrophic Lateral Sclerosis.	
				Posterior Sclerosis (Locomotor Ataxy).	
		D. *Indiscriminate Lesions.*		Diffuse Sclerosis (Friedreich's Disease).	
				Disseminated or Insular Sclerosis.	
				Softening of the Cord (" Myelitis ").	
				Inflammation of the Cord (Myelitis).	
				Intra-Medullary Hæmorrhage.	
				Intra-Medullary Tumour.	
				Ischæmia of the Lumbar Swelling.	
Spinal Paralyses dependent upon Functional Defects.	Intrinsic Causes (Functional)	E. *Functional Defects.*		Toxic Spinal Paralyses.	
				Intermittent Paraplegia.	
				Hysterical Paraplegia.	
				Paraplegia dependent on Idea.	
				Reflex Paraplegia.	

TABLE III.

Relative Acuteness or Chronicity of the several Spinal Affections associated with Paralysis.

Onset Abrupt. (Minutes.)	*Onset Acute.* (Hours or Days.)	*Onset Sub-Acute.* (Weeks.)	*Onset Chronic.* (Months.)	*Onset very Chronic.* (Half-years.)
1. Fractures and Dislocations of Vertebræ. 2. Punctured or Gun-shot Wounds of Spinal Cord.	7. Softening (Thrombotic). 8. Myelitis, Acute. 9. Acute Spinal Paralysis.	16. Sub-acute and Chronic Spinal Paralysis.	18. Cancer of Vertebræ.	24. Lateral Sclerosis (Spasmodic Spinal Paralysis). 25. Amyotrophic Lateral Sclerosis. 26. Locomotor Ataxy. 27. Disseminated Sclerosis. 28. Diffuse Sclerosis (Friedreich's Disease).
3. Concussion of the Spinal Cord. 4. Meningeal Hæmorrhage. 5. Intra-Medullary Hæmorrhage. 6. Ischæmia of the Lumbar Swelling of the Cord.	10. Acute Ascending Paralysis. 11. Toxic Spinal Paralysis. *a.* Lead Palsy. *b.* Alcoholic Paralysis. 12. Intermittent Paraplegia. 13. Hysterical Paraplegia. 14. Paraplegia dependent on Idea. 15. Reflex Paraplegia.	17. Scrofulous Pachymeningitis (with Vertebral Caries).		
			19. Progressive Muscular Atrophy. 20. Pseudo-hypertrophic Paralysis. 21. Cervical Hypertrophic Pachymeningitis. 22. Tumours and Adventitious Products in Spinal Meninges. 23. Tumours in Spinal Cord.	

TABLE IV.

TABULAR DIFFERENTIAL DIAGNOSIS OF SPINAL DISEASES ASSOCIATED WITH PARALYSIS.

(The Numerals in this Table correspond with those placed before the same Affections in Table III., and with the order in which they are subsequently referred to more in detail.)

Group A.		Spinal Paralyses of Abrupt Onset.	1. Fractures and Dislocations of Vertebræ. 2. Punctured or Gun-shot Wounds of Spinal Cord. 3. Concussion of Spinal Cord.	Obviously the result of Traumatisms. (More detailed diagnosis as to Seat and Nature of lesion must be based on general principles, pp. 523-544).
			4. Meningeal Hæmorrhage. 5. Intra-Medullary Hæmorrhage.	For points of differential diagnosis see p. 573.
			6. Ischæmia of the Lumbar Swelling of the Cord.	Associated with Coldness, and absence of pulsation in arteries, of lower extremities. See p. 576.
Group B.	Motor Paralyses mostly associated with *Muscular Atrophy*, and *no loss of Sensibility*. *Pains absent*, or very slight.	Acute or Sub-Acute.	9. Acute Spinal Paralysis.	Area of paralysis diminishing after the first few days—leaving one or both legs most frequently paralysed. See p. 588.
			16. Sub-Acute and Chronic Spinal Paralysis.	Onset more gradual, area of paralysis tending to increase from first for some time. Reaction of Degeneration. See p. 608.
			10. Acute Ascending Paralysis.	Area of paralysis increasing from the first. No loss of knee-jerk and normal electrical reactions. See p. 591.
		Very Chronic or Chronic.	19. Progressive Muscular Atrophy.	Onset very slow, beginning in upper extremities commonly. Atrophy before paralysis. Muscles react to Faradism. See p. 621.
			20. Pseudo-hypertrophic Paralysis.	Disease of childhood. Paresis; tottering gait, with increased size of calves. See p. 624.
			25. Amyotrophic Lateral Sclerosis.	Disease of adult life. Paresis of upper extremities. Twitchings and rigidities of these muscles followed by atrophies. Similar signs in lower extremities with exaggeration of deep reflexes. See p. 640.

Group C.	Chronic Motor Paralyses preceded or associated with *Severe Pains* in limbs or trunk—with or without evidence of coexisting Vertebral Disease.		18. Cancer of Vertebræ.	Secondary to Cancer of Mamma or elsewhere. Disease of middle life or later. Pains very severe. Often rounded prominence of vertebral spines. Legs principally affected. Deep reflexes exaggerated. See p. 615.
			17. Scrofulous Pachymeningitis (with vertebral caries).	Patient of scrofulous type, young or early adult age. Pains less severe—often not spontaneous—but in execution of certain movements, or when spine is jarred. Angular curvature often. Legs most commonly affected, with exaggeration of deep reflexes. See p. 612.
			21. Cervical Hypertrophic Pachymeningitis.	Disease of adult life. Affecting upper extremities first. Severe pains in them, with hyperæsthesia and afterwards anæsthesia. No evidence of cancer or disease of vertebræ. Followed by paralysis of lower extremities. (p. 627)
			26. Locomotor Ataxy.	Lightning pains in lower extremities; absence of knee-jerks. The Argyll-Robertson pupil. Unsteadiness when eyes are closed. Ataxic gait. (p. 642)
			22. Tumours and Adventitious Products in Spinal Meningitis.	By exclusion of other diseases pertaining to Group C. See p. 631.
Group D.	Diseases in which neither *Muscular Atrophies* (early) nor *Severe Pains* are marked features.	Onset Acute.	7. Softening (Thrombotic).	A primary affection. Often sensory as well as motor paralysis. Deep reflexes often exaggerated, not lost as in No. 9. (p. 581)
			8. Acute Myelitis.	A secondary affection usually. Characters clinically very similar to No. 7. (p. 586)
		Onset very Chronic.	27. Disseminated Sclerosis. 28. Diffuse Sclerosis (Friedreich's Disease).	For differential diagnosis, see p. 656.
			24. Primary Lateral Sclerosis.	See p. 638.
			23. Intra-Medullary Tumours.	See p. 634.
		Onset mostly Acute or Sub-Acute.	11. Toxic Spinal Paralyses.	See p. 592.
			12. Intermittent Paraplegia.	See p. 597.
			13. Hysterical Paraplegia.	See p. 598.
			14. Paraplegia Dependent on Idea.	See p. 602.
			15. Reflex Paraplegia.	See p. 604.

SPINAL PARALYSES OF ABRUPT ONSET.

Under this head we have paralyses resulting from the following causes :—

1.—Fractures or Dislocations of Vertebræ.
2.—Punctured or Gun-shot Wounds of Spinal Cord.
3.—Concussion of Spinal Cord.

4.—Meningeal Hæmorrhage.
5.—Intra-Medullary Hæmorrhage.
6.—Ischæmia of the Lumbar Swelling.

These six affections resolve themselves into two groups. 1, 2, and 3 are results of traumatisms, and the primary element in their diagnosis is generally either self-evident or made for us ; 4, and 5 may likewise be results of traumatisms, though they also occur quite independently of any such precursor. The sixth affection is a very rare condition which is altogether independent of traumatic causes.

1. FRACTURES AND DISLOCATIONS OF VERTEBRÆ.

After the occurrence of severe blows or falls, the establishment of the existence of fracture and dislocation (or of dislocation of vertebræ alone) together with the existence of a paraplegic condition, points to the occurrence of a sudden crushing lesion of the spinal cord.

Still, occasionally, there is good reason for believing that a rapid crushing lesion of the spinal cord may take place without the existence of any permanent dislocation. Fracture may have occurred through a vertebra, one half of which may have been driven forward by the original impact to a sufficient extent to crush the cord, though it may almost immediately revert to its proper position.

The level of the lesion in the cord must be determined in accordance with the general principles already set forth (pp. 525-544). Similarly, whether the crush has damaged the whole thickness of the cord at this particular level, or not, is a matter which must be decided by the application of general principles (pp. 523-525).

As a complication we may have, in these cases, to do with **meningeal hæmorrhage** as well as with the crushing lesion. Its positive diagnosis is often extremely difficult in such a case; still some of the characters mentioned further on (p. 573) may guide us.

As further complications or sequelæ, springing up in the course of two or three days, in such crushing lesions of the cord, we may have the supervention of a true **acute myelitis** starting from the injured region, which may or may not be combined with **meningitis.**

This kind of damage to the cord may be produced by the sudden giving-way of a carious vertebra in any part of the spinal column; more rarely from a heavy blow on the back, which does not fracture the spine; or, in a modified form, from the bursting into the spinal canal of an aortic aneurism, after its erosion through the vertebræ. But, in the majority of cases, such wounds of the spinal cord are the results of forms of external violence which cause fracture and dislocation of vertebræ in some portion of the spinal column between the upper cervical and the upper lumbar region.

When this occurs, displacement of vertebræ, even to a slight extent, especially in the dorsal region, in which the spinal canal is narrowest, is sufficient to produce severe pressure upon, or crushing of, the spinal cord. The membranes may not be torn across, but the substance of the cord itself may be greatly compressed or reduced to a blood-stained, semi-fluid mass of pulp. After some hours, there are obvious signs of a commencing inflammatory reaction in the membranes; and above and below the seat of lesion similar changes are apt to be set up in the spinal cord itself, which may go on to the production of a variable amount of inflammatory softening. The patient may die, however, before any of these latter changes have been established.

The **symptoms** in lesions of this kind naturally vary much, according to the region of the cord involved. Still, in spite of differences thus dependent upon the seat of injury, there is a certain general similarity in the symptoms produced by all crushing lesions of the spinal cord. They are usually of this nature:—Complete paralysis, both motor and sensory, of parts below the seat of lesion; in addition to severe pains in the back, girdle pains surrounding the body at the upper limit of sensory and motor paralysis; increased heat or possibly undue coldness of the body throughout the paralysed parts; complete paralysis of bladder with retention of urine, gradually giving place to incontinence; paralysis of rectum, extremely obstinate at first, but subsequently complicated with involuntary evacuations after the administration of purgatives; together with extinction of all reflex actions at first.

In the course of two or three days, if the patient should survive, other general symptoms become well-marked, owing to the establishment of a local meningitis, together with some amount of traumatic myelitis. Among these we have general fever, with an increase of the 'girdle sensation' and of pains in the limbs; twitchings in the limbs or in particular muscles; and also, for a time, a general increase in reflex actions.

The above-mentioned complicating pathological processes may gradually subside, but there will still be danger to life from the supervention of severe cystitis or of extensive bed-sores, together with one or other of the various sequelæ to which such conditions are apt to give rise.

The additional symptoms and variations met with, according as the crushing lesion occurs in different regions of the cord, are as follows. (They will be found to increase in number the higher the lesion is situated in the spinal cord. See also pp. 525-544.)

When it is situated in the *lumbar swelling*, we have, in addition to the limitation of the paralysis to the lower extremities and a more or less complete extinction of related reflex actions, the appearance of rapid atrophy in the paralysed muscles, together with the manifestation of the electrical 'reaction of degeneration.' The bladder and rectum are apt to be completely paralysed.

With the lesion in some part of the *dorsal region* we have sensory and motor paralysis of the trunk up to a certain level, with an absence of the rapid atrophy and before-mentioned electrical reaction in the muscles of the lower extremities, though some atrophy and the presence of this reaction may occur in one or more of the trunk muscles. In addition (and notably with the lesion in higher parts of the dorsal region) there may be some weakness of voice, some interference with the movements of respiration (especially with those of expiration), as well as marked and continuous priapism. The superficial and deep reflexes may be depressed or exalted, according to the degree of completeness of the transverse lesion and the condition of the grey matter below this site.

With the lesion in the *lower cervical region*, the upper extremities are partly paralysed, both as regards sensation and motion; the movements of respiration are much more gravely interfered with (expiration especially), while inspiration is of a purely abdominal type; the voice is notably weak and feeble. Continued erection of the penis is more frequently met with; and in some cases a remarkable hyperpyrexia supervenes, in which the temperature before death may rise to 108°-110° Fahr. Should death not occur in this way, it is very apt to supervene in the course of a few days by gradual failure of respiration, which grows worse than it was in the early days of the affection, owing to the secondary myelitis which becomes established implicating the cord and nerve-roots at a level higher than the original wound. The pulse is often much interfered with, but variously; it may be slower or much more frequent than natural; it may be small, irregular, and frequent; or full, regular, and infrequent in its beats. There may also be signs of paralysis of the vaso-motor nerves supplying the neck and head, perhaps to a more marked extent on one side than on the other.

Where the lesion occurs in the *upper cervical* region of the cord complete paralysis of the trunk and of all four extremities may be recognized, if death does not occur too suddenly to allow even this to be observed. The sudden death, so apt to occur in these cases, is due to the fact that in them the diaphragm is paralysed, as well as the other respiratory muscles. Where the lesion does not involve the whole of the roots of the phrenic nerve, and where the shock has not been too abrupt and violent, life (with extremely difficult respiration and almost complete loss of voice) may be prolonged for a few hours.

An admirable series of cases illustrating these crushing injuries to the spinal cord is to be found in Ollivier's work ('Maladies de la Moelle Epinière,' 3me éd., t. i., p. 253 *et seq.*).

In regard to **diagnosis**, it may be said that if the existence of fracture and dislocation of vertebræ can be substantiated, the probabilities are always in favour of the presence of a crushing lesion in the spinal cord. Otherwise after a very severe fall or blow upon the back, doubts may be at first entertained as to whether we have to do with the effects of concussion alone, or with this *plus* some amount of crushing of the cord or of hæmorrhage upon or beneath its membranes. The subsequent course of the symptoms may, however, in a day or two, enable us to resolve these doubts.

2. PUNCTURED OR GUN-SHOT WOUNDS OF THE SPINAL CORD.

Where such injuries damage the spinal cord and cause paralysis, the longitudinal situation in, as well as the extent of the lesion in the transverse area of, the spinal cord are the principal points requiring to be settled, as in the case of the injuries referred to above.

Similarly, in these cases, there may be the initial complication of a **meningeal hæmorrhage**; while after a time there may be the supervention of **acute myelitis** with or without a **meningitis**.

These wounds often involve only a portion of the transverse area of the cord. It is, indeed, in this class of cases more especially that *hemiplegia spinalis* and *hemiparaplegia* are met with. Thus, where a unilateral lesion exists in the mid or upper cervical region, both arm and leg are paralysed, so that the state known as *hemiplegia spinalis* is produced; but where it occurs in the dorsal region, the one leg only is paralysed, and we have what is known as *hemiparaplegia*.

The essential peculiarity in these latter cases is that on the side of lesion there is complete motor paralysis in the limbs or limb below; while on the opposite side, the limbs or limb, and the trunk up to the middle line, are more or less completely anæsthetic—sensitiveness to impressions of touch, pain, temperature, and tickling being alike abolished.

Other minor peculiarities are these :—On the side of *motor paralysis*, there is also vaso-motor paralysis, which carries with it, as consequences, (*a*) an elevation of temperature (from 1½° to 2° F.), and (*b*) a hyperæsthesia for all modes of sensibility (owing in part to hyperæmia in the limb and cord). Surrounding the body, at the level of the upper margin of anæsthesia on the side of sensory defect, there is usually a narrow girdle of hyperæsthesia; whilst below this level, on the side of the lesion, there is a half-band of hemianæsthesia—whose depth varies with the longitudinal extent of the lesion. (The complete zone of hyperæsthesia is probably due to hyperæmia of nerve-roots, and of the grey matter of the cord immediately above the lesion; while the half-zone of anæsthesia is dependent upon destruction of the sensory nerve-roots, and of the spinal cord for a certain extent.)

If bed-sores occur, they are met with on the side of sensory paralysis; while, in one or two cases, signs of a joint-affection (in the knee principally) have occurred on the side of motor paralysis. There seems no reason for expecting any special muscular atrophy or diminution of faradic irritability on the side of motor paralysis, except in those muscles whose nerve-supply comes from the portion of the anterior cornua actually destroyed by the lesion. In many cases, especially at first, there is paralysis of the bladder and of the rectum, or there may be incontinence of urine. Later on, these troubles tend to diminish. Nothing definite is known in regard to the condition of the skin-reflexes and tendon-reflexes in these states.

Where anomalies exist in regard to the extent of decussation of the pyramids (Flechsig), the above-described effects of unilateral lesions of the cord would also undergo corresponding variations.

The primary cause of the patient's condition is generally only too obvious. It may be clear that we have to do either with a punctured or with a gun-shot wound in some region of the spine; but subsequently many, and often very difficult, questions require to be solved. It is of first importance to learn whether the cord itself is really damaged, or whether the symptoms are in the main caused by epi-dural or sub-arachnoid hæmorrhages. In the former case there will be evidence of complete or partial interruption of conduction in the cord, to or from all parts below the seat of lesion, and not of a mere local implication of nerve-roots. Thus if it seem probable that the cord itself is damaged, we have to determine whether it is completely cut across, or only partially damaged—and, if the latter, to what extent. These questions must be decided in the main by reference to the signs cited on pp. 523-525.

Should the patient be seen for the first time several days after the injury, an exact diagnosis as to the amount of damage to the cord itself is often greatly obscured by the existence then of certain secondary pathological conditions—more especially localised inflammation of the meninges, or secondary inflammatory softening, extending perhaps above or below, or on both directions, from the original wound. A process of softening may also extend transversely through the whole substance of the cord, even where only a unilateral lesion had previously existed.

3. CONCUSSION OF THE SPINAL CORD.

Here, again, the primary or initial diagnosis is usually made for us. We are called to see a person who has fallen from a height, or one who has received some severe blow on the back, or a severe general shaking, such as may occur in a railway accident.

Such cases require however a most careful study before any positive diagnosis can be arrived at.

We must bear in mind, in the first place, that some of the symptoms presented by the patient may be due to concussion of the brain coexisting with concussion of the spinal cord. The same accident may easily have caused concussion of both these parts of the nervous system.

Then, again, the question must always arise whether we have to do with something more than concussion or not. In mere concussion of the spinal cord there should be no discoverable lesion. This however is far from being always the case. Concussion of the cord may be complicated by the occurrence of small extravasations of blood into its substance, or by actual ruptures of the nerve tissue, especially of the grey matter. Both Gull and myself have recorded cases of this kind—cases too in which there was no external or visible injury, and where a paraplegic condition was induced immediately after the fall that determined the concussion and the lesion in the cord.

Another cause of complication is the occurrence of a meningeal hæmorrhage at the time of the blow or fall.

Finally within a day or two (and we may not see the patient till after such an interval has elapsed), there is the possibility of some local and sub-acute inflammation being set up in the membranes of the cord.

In the great majority of these cases no complete paralysis is induced, even at first. There may, at most, be paresis of one or more limbs, general prostration, nausea with occasional vomiting, a rapid and possibly irregular or intermittent pulse (especially after the least exertion), with occasional startings and twitchings of the limbs, whose sensibility may be diminished, exalted, or unaffected. The temperature will probably be at first depressed, as a result of shock, though subsequently a febrile elevation may continue for some days. The tongue may be furred, the appetite bad, the bowels constipated; while in regard to micturition there may be either some delay and difficulty, or, on the contrary, an irritability of the bladder, with difficulty in retaining its contents after the desire to micturate is once felt. With this there is often general restlessness, nervousness, and insomnia.

In more severe cases of concussion, even where there is no complication resulting from appreciable lesions, the shock to the system may be more profound, and there may be paralysis of limbs lasting, perhaps, for some days and then rather suddenly disappearing.

The questions to be determined are, whether, looking to the symptoms presented by the patient, there is likely to be any organic lesion or change in the spinal cord or its membranes; or whether we have to do with mere functional perturbations induced by the shock or blow to which the patient has been subjected. In the absence of definite paralysis, or even with its presence for the first few days, the answer to this preliminary question will often be shrouded in doubt. To come to a definite opinion as to the precise nature of the change which a spinal cord, deemed to be damaged in some way after a concussion, has undergone, lapse of time and several examinations of the patient are often required.

In many cases in which compensation for an injury is claimed a further complication appears. Here it is that the difficulty arises as to how much the symptoms experienced, or said to be experienced, may be due to an excited imagination, and how much to causes independent of the imagination, whether voluntarily or involuntarily aroused. It must be conceded that symptoms of injury are undoubtedly feigned by unscrupulous persons ; and it seems also equally clear that, even unknowingly to the patient, the excitement consequent upon the accident, the details heard concerning the injuries of others, combined with the inquiries of doctors and of sympathising friends, tend to keep up and to exaggerate symptoms, in many nervous patients, over and above those that may have resulted from the shock. Such patients, also, may make a more speedy recovery, subsequent to trial and compensation, than they had been making before the trial, and yet they *may* not have been in any sense impostors. It is true that such persons, however, do not recover quite so quickly as those others who, for their own unscrupulous ends, have been previously exciting their imaginations in a voluntary manner.

4. MENINGEAL HÆMORRHAGE, AND 5. INTRA-MEDULLARY HÆMORRHAGE.

Each of these conditions will, of course, produce different sets of symptoms according to the amount of blood effused, and according to the particular region of the cord (that is, the particular longitudinal and transverse region) that happens to be pressed upon or structurally damaged.

Apart from such individual differences, which in each case have to be interpreted in accordance with the general data previously set forth, generic differences of a marked character are often found to exist between the two sets of cases. This may be seen from a comparison of their subjoined respective characteristics.

Meningeal Hæmorrhage.	**Intra-Medullary Hæmorrhage.**
Not uncommon in association with traumatisms, and violent muscular or respiratory spasms—occasionally during the lifting of very heavy weights.	Rare, and slight in amount, in association with traumatisms; and very rare as a primary pathological accident.
May occur at any age.	Primary hæmorrhage most common between the ages of twenty and forty.
Abrupt pain in the back shooting into the limbs, which may be succeeded by hyperæsthesia.	Abrupt momentary pain in the back, possibly felt also in the limbs.
Paralysis and anæsthesia generally slight and not lasting very long. Anæsthesia may be absent altogether. Spasms and rigidities are common.	Well marked paralysis and profound anæsthesia in the paralysed parts, from the first. Spasms and rigidities are rare.
No tendency to the occurrence of bedsores.	Bedsores are particularly apt to occur.
Paralysis of sphincters rare; no tendency for cystitis to occur.	Paralysis of sphincters common; cystitis is very prone to occur.
Complete recovery often occurs.	Very frequently proves fatal; recovery never complete.

These symptoms of **meningeal hæmorrhages** are in many cases rather vague and ill-defined. They will vary much in distinctness according to the amount and rapidity with which the blood is poured out.

Again, in traumatic cases, the symptoms may be much obscured by the nature of the causal conditions, and by the existence of lesions elsewhere. Thus, where a meningeal effusion occurs in the cervical region in a traumatic case in which there is obvious head injury with a condition of stupor, it is almost certain not to be diagnosed. The patient is not sensible enough to complain of pain; and the irregular respiration and small disordered pulse, with slight tremor or rigidity of one or both upper extremities, may, with more probability, be ascribed to multiple head lesions—as actually happened in a case which came under my care a few years ago.

It may be almost impossible, again, to diagnose hæmorrhage into the spinal meninges in cases where it occurs as a concomitant of other grave diseases—such as tetanus, eclampsia, or cerebral hæmorrhage; and also in cases where it merely complicates a traumatic injury of the spinal cord itself.

In other cases, the presence of certain causal conditions, together with the abrupt commencement of spinal symptoms in such combinations as have been above given, is sufficient to enable us to diagnose a meningeal hæmorrhage from hæmorrhage into the substance of the cord, as well as from acute thrombotic softening.

The gradual onset of the symptoms arising from tumours of the spinal cord or of the spinal meninges makes it comparatively easy to separate these affections from meningeal hæmorrhages.

Hæmorrhage into the Spinal Cord is a comparatively rare event. It occurs under three different conditions, namely:—(1) as a result of concussion or violence; (2) as a secondary event, consequent upon a definite pre-existing morbid condition; and (3) as a primary event, or local pathological accident.

We are here specially concerned with hæmorrhages into the spinal cord belonging to the third of these categories, and may in a few words dismiss the other two.

(1). Traumatic hæmorrhages, small in extent, may, as already stated, occur in almost any region or part of the cord as a result of some severe concussion (p. 571). Again, it may occur in the grey matter, and even in the white substance to a smaller extent, close to, and as appanage of, wounds of the cord. In each of these cases, symptoms due to the hæmorrhage itself would probably be obscured by the general set of symptoms resulting from the concussion or injury.

(2). Secondary hæmorrhages are, however, more closely connected, from the point of view of symptomatology, with those forming the special subject of this section. During the growth of certain soft tumours in the cord, a rupture of some of their vessels may take place, so as to cause hæmorrhage either into the growth itself, or else into contiguous regions of the cord. Such an event would be signalised clinically by the sudden exacerbation of the symptoms previously existing. But a combination of greater importance, though one of considerable obscurity, consists in the coexistence of a 'central myelitis' of the grey matter of the cord through more or less of its extent, together with a central hæmorrhage of nearly similar extent. The existence of any such 'central myelitis' as an independent disease of the cord seems to the writer very doubtful. It is at least equally probable that the hæmorrhage has been primary, and that the 'myelitis' or softening is of secondary origin around the blood-clot. It need not be denied, of course, that in other cases hæmorrhage does occur occasionally into the midst of a focus of softened tissue in the spinal cord, just as it occurs occasionally under similar conditions in the midst of softened brain-tissue.

(3). Primary hæmorrhages differ as regards the amount, the site, and the distribution of the blood effused in different cases. In connection with scorbutic states, and also independently of these, small hæmorrhages may occasionally occur into the substance of the cord, without producing any distinct symptoms. But, at other times, a comparatively large quantity of blood may be effused into the cord, and then it occurs almost invariably into the grey matter in the central regions of the cord, through which it may extend for a variable distance. When the quantity is smaller, the blood may be effused into the grey matter of one side only.

Where the hæmorrhage invades pretty fully, but is limited to, the grey matter of one half of the cord, we may have groups of symptoms that take the form of *hemiplegia spinalis* or *hemiparaplegia*. (See also p. 569.)

From the point of view of **diagnosis**, it may be said that the absolutely sudden onset of the paralysis, which may be complete in the lower extremities in the course of a few minutes (especially when associated with a sudden painful sensation in the back, or one which radiates into the limbs) ; as well as the almost complete and sudden loss or great impairment of sensibility in the paralysed parts, form a group of symptoms which is typically distinctive of hæmorrhage into the grey matter of the cord.

It does, however, happen occasionally that a process of softening—probably caused by thrombosis—has its occasioning conditions initiated suddenly. When this occurs, paraplegia sets in almost as abruptly as if it were occasioned by hæmorrhage; but then it is usually an incomplete paraplegia, and, for a time at least, unaccompanied by loss of sensibility. In the course of a few days, in such a case, sensory paralysis may supervene, and the motor paralysis becomes more complete (p. 582). In the exceptional cases of paraplegia of sudden onset due to this cause, there is generally no initial pain in the back, though I have known initial pains and burning sensations in the limbs to be complained of.

6. ISCHÆMIA OF THE LUMBAR SWELLING OF THE CORD.

Exceptional cases occur occasionally in which a condition of real anæmia of the spinal cord is brought about in man, just as it has been brought about in one of the lower animals whose abdominal aorta has been tied or compressed. When the blood-supply is thus suddenly cut off from the lumbar region of the cord in animals, their hinder limbs become paralysed almost immediately, and continue paralysed as long as the blood-supply of the cord happens to be arrested. But if, after a mere brief interval, the blood is again allowed to take its natural course, the temporary paralysis disappears completely in a very short time.

A condition of this kind seems to have occurred in a patient, formerly under the care of Sir W. Gull, who suddenly became paraplegic, apparently owing to an abrupt arrest of the blood-current through the abdominal aorta, as was indicated by the cessation of the femoral and other pulses in the lower extremities ('Guy's Hospital Reports,' 1857, p. 311). The man continued paraplegic for months, and only recovered when the collateral circulation became, after a time, pretty fully established.

In a very few other cases referred to by Erb, in which paraplegic symptoms were associated with an obstruction of some kind in the abdominal aorta, he thinks that these symptoms, supervening as they did rather less suddenly, may have been in great part due to the deficient blood-supply to the muscles and nerves of the lower extremities, rather than to anæmia of the cord—to a peripheral, that is, rather than to a centric anæmia—such as may occur when the cauda equina, rather than the lumbar swelling of the cord, is deprived of its blood supply.

These extremely rare cases of paraplegia due to **compression, thrombosis or embolism of the abdominal aorta,** if they are ever met with, may be diagnosed by the sudden supervention of paraplegia, in which there is both motor and sensory paralysis of the lower extremities;

an abolition of all reflex acts dependent upon the lower part of the spinal cord; paralysis of the bladder and rectum; and also the simultaneous disappearance of the femoral and other pulses in the lower extremities. These symptoms are followed speedily by coldness and œdema of the feet and legs—signs which may subsequently disappear gradually (together with the paralysis), if, after a time, the compression or obstruction be removed, or whenever a collateral circulation becomes established.

SPINAL PARALYSES OF ACUTE ONSET.

Under this head we have paralysis resulting from the following causes:—

7. Softening of the Spinal Cord (thrombotic).
8. Acute Myelitis—rarely primary.
9. Acute Spinal Paralysis.
10. Acute Ascending Paralysis.
11. Toxic Spinal Paralysis.
12. Intermittent Paraplegia.
13. Hysterical Paraplegia.
14. Paraplegia dependent upon Idea.
15. Reflex Paraplegia.

So-called Functional Diseases of the Spinal Cord.

The forms of Paralysis Nos. 10-15 *inclusive, may also, at times, have a Sub-Acute Onset (see Table* III., *p.* 562*).*

7. SOFTENING OF THE SPINAL CORD (THROMBOTIC).

The notion that common 'softenings' of the spinal cord are of inflammatory origin has persisted with little alteration, although for nearly twenty years pathologists have been interpreting altogether differently the mode of production of apparently similar 'softenings' of the cerebrum and cerebellum. Can it be that 'softening' as it occurs in the majority of cases in these latter organs is of non-inflammatory origin; while in the majority of apparently similar cases occurring in the spinal cord, the process is really inflammatory in its nature?

We have already pointed out that embolism of spinal arteries is a very rare event, and that, when it does occur, it can only be in very small vessels, whose occlusion would merely give rise to minute foci of softening not capable of causing any distinct paralytic condition.

We must look, therefore, to thrombosis rather than embolism, as the common cause of primary softening of the spinal cord.

I have already intimated my opinion (p. 552) that far too large a share is assigned to inflammation in the pathogenesis of diseases of the spinal cord. This mistake is particularly obvious in regard to acute inflammations. It has long been the fashion to speak of almost every focus of 'softening' that occurs in the spinal cord as being the result of an 'acute myelitis'; and we find even Erb (Ziemssen's 'Cyclopædia,' vol. xiii., p. 470) putting forward, as characteristics of an inflammatory softening, peculiarities which ought not to be regarded in such a light—and this although he seems otherwise strongly inclined to hold an opinion very similar to that above expressed. While admitting that a true myelitis is not distinguishable macroscopically, in the great majority of cases, from a simple or non-inflammatory softening, Erb adds a statement to the effect that the 'microscopical examination can alone furnish conclusive evidence.' In the opinion of the writer, however, such evidence as that which is cited by Erb is quite inconclusive.

It is evident, indeed, that we are still almost as destitute of microscopical as we are of macroscopical characters, of a trustworthy description, for enabling us to decide whether any given focus of softening has been of inflammatory or of simple non-inflammatory origin. Such researches as those of Hamilton ('Quart. Journ. of Micros. Science,' Oct., 1875) and others must be prosecuted further, and multiplied, before any certain means of deciding this question will exist.

In the present state of knowledge, therefore, it would appear that 'non-inflammatory softenings' of the cord are represented by the primary and apparently idiopathic 'softenings' which frequently occur in this organ.

In regard to their distribution or extent in the cord many varieties of softening exist. These have been commonly recognized, though they have been mostly described under corresponding designations as so many varieties of 'myelitis.' Thus, we may have a 'complete or total transverse softening,' involving the entire thickness of the cord for a variable longitudinal extent, either in the lumbar, the dorsal, or in the cervical region. Or the softening may be more limited to certain subdivisions of the cord in one or other of these regions—and then constitute an 'incomplete transverse softening.' Thus it may, in one set of cases, principally affect the anterior columns and grey matter; in another set, the posterior columns and more or less of the grey matter. Or the softening may be central, and almost confined to the grey matter through a considerable extent of the cord, as in 'diffuse central softening'; when this change involves the white columns as well as the grey matter for a considerable extent, we have what is called 'diffuse softening' of the cord. When a small focus of softening exists which only involves part of the transverse area of the cord, and that for a very limited extent, we have a 'circumscribed softening' of the cord; and where many of these small foci are scattered through different parts and regions of the organ, we have what is known as 'disseminated softening.'

The symptomatology of this disease presents an extremely wide range, in accordance with the varying extent and sites of the foci of softening that may exist in different patients.

In 'circumscribed' and 'disseminated softening,' for instance, the symptomatology would be excessively variable in different patients, and, in the latter class of cases, it would be generally extremely difficult to arrive at a diagnosis. The symptoms could, in fact, only be interpreted by the light of the general principles applicable to the regional and pathological diagnosis of lesions in the cord.

Again, in cases of 'diffuse central softening' the symptoms—except for the fact that they set in gradually rather than abruptly—would bear a close resemblance to those of hæmorrhage into the spinal cord, in which the blood is effused into the central grey matter for a certain extent (p. 574). There is some doubt, indeed, whether these latter cases may not occur principally as epiphenomena sequential to a primary central softening.

The symptomatology of 'incomplete transverse softening' of the cord, is, for the most part, exemplified by the second stages of various forms of so-called 'compression myelitis'—cases, that is, in which, in one set of cases, the anterior regions of the cord more especially are principally pressed upon either by tumours, or by the inflammatory products associated with vertebral caries ('Pott's Disease'); while in another set, the posterior columns and posterior grey matter may undergo a similar softening, under the influence of the pressure of a new-growth impinging upon the cord from behind. Cases of this type, however, may easily, and do often, merge into 'complete transverse softening' (commonly known as 'complete transverse myelitis'). Both complete and incomplete forms of softening often occur, moreover, in the cord, quite independently of pressure.

Of these states it will be well, for the sake of brevity, to confine our attention principally to 'complete transverse softening.'

In a case of **complete transverse softening** involving the mid-dorsal region, the temperature in the axilla usually varies between 98° and 100° F., though with an extension of the pathological process, or towards the close of the disease, it may rise to 101°, 102°, or even higher. Meanwhile, the lower extremities themselves are often distinctly cold to the hand—the temperature being, in some cases, more or less subnormal. It is important to note this, because it might have been supposed that hyperæmia and a slightly elevated temperature would exist, owing to the vaso-motor nerves of the limbs being paralysed.

The motor paralysis of the lower extremities is absolute, and the abdominal muscles are also powerless. The feet, as the patient lies in bed, are extended and often inverted, so that the great toes cross one another. The skin after a time tends to become dry and scurfy. The muscles feel flabby to the hand, but they waste only to a slight extent, and continue week after week to show only a small amount, if any, of diminution in the degree of their irritability to faradic and to galvanic currents.

The sensibility of the limbs is completely abolished both for tactile and painful impressions, as well as for differences of temperature and tickling. A like abolition of sensibility exists over the trunk up to the level of the 'ensiform area' while above this level the sensibility becomes quite natural. Though the upper limit of anæsthesia may be quite sharply defined, yet, in these cases of complete transverse softening, there is often no distinct 'girdle-sensation.'

The muscles of the lower extremities may show some slight irritability when they are forcibly tapped, and when the soles of the feet are strongly tickled there may be very slight movements of the toes; but beyond this there is generally an entire absence of all reflex movements—there is no ankle-clonus, no knee-reflex, and a similar absence of the cremasteric and abdominal reflexes.* In the initial stages of the affection, however, and especially when the softening does not involve the whole transverse area of the cord, all these reflexes may be extremely well-marked for a time, though they tend gradually to diminish (see p. 538).

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* In one recent case in which paraplegia had existed for over three months in consequence of a complete transverse softening in the upper dorsal region (with the above-mentioned clinical signs), the writer was much struck with the extremely pallid appearance of the grey matter through the whole length of the cord below the seat of softening. The absence of the reflexes may be in part due to such condition of the grey matter, and this itself may be caused by a spasm of its vessels in some way induced by the lesion above. Some amount of spasm may also exist in the vessels of the limbs, whose temperature is often rather subnormal.

For the first ten days or a fortnight there is often complete retention of urine, but after this time, when the lumbar region of the cord again becomes capable of manifesting to some extent its centric functions, the initial retention gives place to incontinence of urine. This fluid may then be discharged at intervals of from two to three hours in small quantities, owing to the occurrence of reflex contractions of the bladder whenever it attains a certain degree of fulness. The passage of a catheter, however, in these cases will often show that the bladder is never completely emptied—two to four ounces remaining after the reflex contractions. Unless special precautions are taken, the urine, in such patients, speedily becomes ammoniacal, and more or less loaded with mucus.

The bowels are usually constipated, and relieved only after the administration of aperients or enemata. At these times there is generally incontinence of fæces—the patient having no power of controlling the reflex actions concerned in defæcation when they have once been strongly excited. The actual passage of the motion is, moreover, unfelt.

Under the irritative influences emanating from the seat of softening during the period of its establishment, a small bed-sore may begin to form, often amenable to treatment. Later on, sloughs are apt to form upon the heels, over the malleoli, and in other situations habitually exposed to continuous pressure. But the most frequent site for intractable sloughing bed-sores is over the sacrum. Inflammation of the mucous membrane of the bladder is at last set up ; and the inflammation may extend up one or both ureters, so as to implicate the pelvis of the kidney, when minute abscesses may also form in the kidney itself.

Under the influence of these various conditions the patient's appetite and strength gradually fail ; emaciation proceeds ; and death after a time may come from sheer exhaustion, aided, perhaps, by some intercurrent inflammatory affection of the lungs.

In regard to **diagnosis**, it may be said that the recognition of this disease at the bedside often presents considerable difficulties. We must be guided partly (*a*) by the patient's history and state; partly (*b*) by the mode of onset of the disease; and partly (*c*) by the symptoms of the fully established affection.

(*a*) The points in regard to the previous history which are of principal significance are these. The disease presents itself as a spontaneous or idiopathic affection, sometimes without apparent cause or definite antecedent conditions of any kind, but sometimes as a sequence of one or other of various known and common antecedents.

Thus, in certain cases, the symptoms set in more or less suddenly after some great bodily fatigue; in others after extreme sexual excesses; or they may occur during the period of convalescence from certain acute fevers, such as variola, typhus, and other exanthemata, or after rheumatic fever. During the first week or two after childbirth there is likewise the possibility of the initiation of such symptoms; and also in the later stages of syphilis. These different conditions may act very variously in contributing to bring about a focus of softening in the spinal cord, and nothing more than conjectures can be advanced in regard to its actual pathogenesis in the several cases.

Again, the symptoms indicative of a primary softening of the cord may set in after the action of other conditions, regarded by some as 'exciting' rather than as 'predisposing causes.' Of these the following may be enumerated :—Prolonged exposure to cold and wet; sudden suppression of the menses or of other accustomed fluxes; violent emotional disturbances; or the existence of some inflammation in one or other of the pelvic organs, such as the uterus or the bladder and urethra (instances of the latter class being some of the cases formerly supposed to be paralyses of 'reflex' origin). In regard to the mode of operation of these 'exciting causes,' all that is certainly known is, that softening of the cord seems to set in not unfrequently in persons who have been subjected to one or other of them.

In regard to (*b*) the mode of onset, this is usually not abrupt and sudden; there is often rather a slow increase of paralysis during a week, ten days, or a fortnight. Still, it is a fact that softening of the cord (apparently due to thrombosis) does occasionally cause a sudden incomplete paralysis, though such paralysis increases subsequently in the manner above stated. Such a case must not therefore be confounded with hæmorrhage into the cord, merely by reason of its absolutely abrupt onset.

The extent to which the diagnosis turns upon (c) the nature of the symptoms of the fully-established affection cannot be very definitely defined except in some cases. When the softening is slight and partial, it gives rise to no distinctive symptoms; but where there are clinical signs of the existence of a complete transverse lesion, the chances are that the lesion itself, if not a primary, is at all events a secondary softening.

In regard to the regional diagnosis in cases of softening of the spinal cord, the following points require to be borne in mind:—

The indications as to the transverse area involved, and as to the upper limits of the change in the spinal cord, are wholly derivable from the presence or absence of the various signs and symptoms which have already been set forth (pp. 523-524).

The attempt to ascertain the lower level of the lesion, and consequently its longitudinal extent in the cord, is always difficult, and often cannot be achieved with any success. The indications are all obscure, uncertain, and apt to fail. This is especially the case if we attempt to base an opinion on the fact of the existence or absence of superficial or deep reflexes (see p. 537). Thus, complete transverse softening may exist in the upper dorsal region, and extensive secondary degenerations may have been produced, yet for week after week there may be a complete absence of all the reflexes (superficial and deep) dependent upon the cord below the upper dorsal region. This the writer has ascertained by repeated clinical examinations of cases whose nature has been subsequently verified post-mortem.

8. ACUTE MYELITIS.

By many writers and practitioners the term 'myelitis' is used in a somewhat vague manner, and with an evident tendency to include under it as many different affections or states of the spinal cord as possible. Critical discrimination seems to have been, and still seems to be, somewhat in abeyance by many who describe or report cases of disease of the spinal cord. They set down, only too frequently, as instances of 'myelitis' not only all cases in which the substance of the spinal cord is softened, but still more all those in which it is indurated—and at times, no less impartially, those in which it is merely degenerated.

The various states of the cord to which the term 'myelitis' has been principally applied, may be ranged under four categories.

(1).—Softenings of the Cord are the conditions which, as we have already pointed out, are by almost all observers and writers regarded as instances of 'Acute Myelitis.' Yet to a large majority of these conditions any such name is altogether inappropriate. The special subject of this section, however, will be those forms of softening to which the name 'myelitis' may be more appropriately applied.

(2).—Next come the cases of spinal disease that commonly go by the name of 'anterior polio-myelitis,' 'cornual myelitis,' or 'acute spinal paralysis.' Of late something like definite evidence concerning the inflammatory nature of the initial morbid conditions has been forthcoming, although in its later stages (that is, at those stages with which alone we had previous familiarity) the morbid process bears rather the impress of a degenerative condition. Of this, however, more will be said in the next section.

Just as there are the two foregoing sets of acute processes which are commonly included under the term 'acute myelitis,' so are there two sets of chronic processes, to which the term 'chronic myelitis' is commonly applied.

(3).—'Secondary degenerations' of the spinal cord have, indeed, in spite of their name, and of what is known as to their origin, been erroneously regarded of late by some writers as inflammatory changes (Ziemssen's 'Cyclopædia,' vol. xiii., p. 769), that is, forms of 'chronic myelitis.' In this condition we have bands of tissue-change produced, in which some of the characteristics of non-inflammatory softening are blended with those pertaining to a patch of sclerosis. In brief, we have effects resulting from a primary fatty degeneration of the nerve-fibres, and a secondary hyperplasia of the neuroglia; and from first to last there is not the least reason for believing in the existence of an inflammatory process (see pp. 201, and 553).

(4).—If we turn now to 'scleroses' of the cord of primary origin, we again meet with processes which are commonly regarded and described as forms of 'chronic myelitis.' This nomenclature is objectionable as applied to the processes in the spinal cord, just as it is in its application to like processes occurring in other organs, as the liver, the lungs, or the kidneys. Fibroid overgrowth, which forms the basis of so many examples of 'cirrhosis' or 'sclerosis' in different organs and tissues of the body, is a process pathologically intermediate between inflammation, on the one hand, and degeneration on the other. Thus, what were formerly named 'interstitial inflammations,' are now the 'non-inflammatory hyperplasias' of some pathologists, and the 'fibroid degenerations' of others. It would seem that the view as to the inflammatory nature of such processes is erroneous, if we look either to what is known concerning their modes of initiation, or to the actual nature of the changes themselves (which agree in almost every particular with those of infiltrating new growths); it would seem, moreover, not less erroneous if we look to the clinical history of the affections themselves in which these scleroses occur. It conveys, therefore, an altogether erroneous implication to speak of such mere fibroid overgrowths or indurations as so many instances of 'chronic myelitis.'

It will be seen that the writer attributes to inflammation a far more restricted *rôle* in the production of morbid conditions of the spinal cord than is customary.

That many of the forms of 'softening' of the spinal cord are of a simply degenerative type (due to disturbances of blood-supply), and that, in the great majority of cases, these are the instances in which 'softening' appears to occur as a primary process, the writer feels assured. On the other hand, it seems clear that, in many cases, changes, truly inflammatory in their origin and progress, may terminate in the production of states of 'softening' of the cord, which are indistinguishable by naked eye from the softenings of degenerative type, and which can as yet also be very imperfectly discriminated by the microscope.

These latter true **inflammatory softenings** very rarely occur as primary pathological states; they are met with rather as secondary changes, after one or other of the following primary lesions or morbid conditions.

Thus we may get inflammatory softenings spreading (*a*) around and from wounds or other traumatic lesions of the spinal cord; or (*b*) starting from some blood-clot or tumour situated in, or pressing upon, the substance of the cord. It is not by any means clear, however, that all the forms of softening which arise in the latter manner should be regarded as of an inflammatory nature; and much room for doubt also exists as to the real pathogenesis of many cases of so-called 'compression myelitis' (p. 609).

Another cause of true inflammatory changes in the spinal cord ('myelitis peripherica') is to be found (*c*) in spinal leptomeningitis.

Suppuration is clearly a process of inflammatory origin, and might therefore be expected to occur occasionally in the midst of 'softenings' which result from inflammation. In the light of what has been said above, the following statement by Erb is of considerable interest. He says:—"Actual suppuration occurs very rarely in acute myelitis. When abscess of the cord does form, it is generally secondary to a severe traumatic lesion or to suppurative meningitis. In spontaneous myelitis, on the other hand, suppuration is exceedingly rare, and has only been observed in a very few cases." Thus, suppuration is met with just in those forms of softening ('myelitis') which are undoubtedly of inflammatory origin; and, on the other hand, it is not met with in the ordinary cases of primary or spontaneous softening, here assumed to be of non-inflammatory nature.

In instances other than those above mentioned, suppuration only rarely occurs in the spinal cord. Small disseminated abscesses may, however, be found in pyæmic cases, as they are in the brain and in other organs.

One other condition requires to be referred to here, and that is the so-called **acute central myelitis**, described originally by Albers, and afterwards studied by Hayem ('Archives de Physiologie,' 1874, p. 603). These are cases in which apparently spontaneous 'softening' is met with, implicating in the main the central grey matter, and that often through a considerable extent of the cord. At times, however, the softening extends beyond the grey matter, so as to involve more or less of the surrounding white substance, when it has been termed myelitis diffusa. Considerable obscurity still prevails in regard to the ætiology of these affections. In some cases, such a change has been met with as part of an infective process, in which minute vessels in the grey matter of the cord have been found obstructed with micrococci. Occasionally, moreover, in certain, at present, imperfectly known conditions, minute thromboses may, as Hamilton has shown, occur throughout the spinal cord, and more especially in its grey matter, and thus lead on, in the main, to the production of a central softening ('Brit. and For. Med. Chir. Review,' April, 1876, p. 447). In this latter case, the patient was suffering from pyelitis, and it is supposed that there may have been some blood-poisoning. Still it was not ascertained that the multitudes of minute thrombi were either associated with or caused by micrococci in the vessels. It appears probable, however, that if, from any cause, minute widespread obstructions of small vessels occur in the spinal cord, softening would take place principally in the grey matter, owing to its greater vascularity. We should thus get that particular distribution of this change which is met with more especially in cases of so-called 'acute central' or 'diffuse myelitis.'

A careful study of the two cases of this disease recorded by Hayem has by no means sufficed to convince me that they ought to be regarded as having had an inflammatory origin. Neither the symptoms nor the mode of onset of the disease lend any distinct support to this view; nor do the results of the elaborate examination to which the spinal cords were submitted by this accomplished observer, show, at all conclusively, that the pathological conditions met with were inflammatory either at their commencement or in their subsequent progress.

From what has been said it will be gathered that, in the writer's opinion, true inflammatory conditions of the cord are only with extreme rarity of primary origin, and that they occur, for the most part, as secondary complications in association (*a*) with wounds or injuries of the cord; (*b*) with foreign bodies in its substance; or (*c*) with spinal leptomeningitis, either simple or tubercular.

The supervention of a real myelitis in the course of either of these diseases of the spinal cord would perhaps be associated with an exaggeration of the already existing febrile condition; with an increase in the amount of paralysis, and in the degree of interference with sensibility; possibly also with more pain, restlessness, and spasms.

From the point of view of **diagnosis** this is about all that can be said at present.

Acute myelitis may, as already indicated, become associated with more or less of distinct suppuration, and almost certainly goes on to the formation of well-marked foci of softening. These may remain limited in site, but occasionally they have a distinct tendency to spread above and below the original seat of injury or disease.

9. ACUTE SPINAL PARALYSIS.

This affection occurs commonly in children, but more rarely in adults.

Its other names 'anterior polio-myelitis' and 'anterior cornual myelitis' imply that it is an affection of inflammatory origin. Until quite recently no distinct evidence could be adduced in proof of this position, partly because no thorough post-mortem examination of the cord had been made in cases in which death had occurred at a period not very remote from the onset of the disease. Lately, this kind of evidence has been forthcoming, and with the result of decidedly strengthening the view that the primary condition is an inflammatory process, which localises itself in the anterior cornua of the cord, and mainly in the lumbar and cervical regions. In later stages of the disease, the lesion met with is a wasted condition of the parts of the anterior cornua involved, the great ganglion cells therein being notably atrophied, and the surrounding neuroglia more or less overgrown.

But whether we look upon the initial pathological condition as an inflammation limited to the regions of the anterior cornua, or as a peculiar atrophic degeneration primarily involving the great ganglion cells of the anterior cornua, it is almost equally difficult for us to give any rational explanation of the conditions which immediately determine this or that localised process.

The characteristics of this affection are so distinct and well defined, as to make it, in the majority of cases, one of the easiest of all the diseases of the spinal cord to diagnose.

There may be slight initial febrile symptoms, accompanied by some headache, sickness, and, on rare occasions, convulsions when the patient is a child. The latter symptoms are usually absent in adults. Such a condition may supervene abruptly in the midst of apparent health. From the fact that it was originally only known to occur in young children, this affection was formerly commonly known as 'infantile paralysis.'

Paralysis then sets in speedily—it may be within a few hours—and is more or less widespread. The muscles are flaccid; reflex actions (except, it may be, the plantar reflex) are abolished or greatly diminished. In the course of a few days, generally, some improvement as regards motor power sets in in certain parts, and very slowly progresses in them. It may go on, continuously, to complete recovery in the course of a few months; or, as often happens, such recovery is only partial. In the latter case, certain muscles or groups of muscles remain paralysed, and in them a rapid atrophy occurs. When tested electrically, these muscles exhibit the 'reaction of degeneration' (p. 10). The affected parts are not at first notably altered in temperature, but, after a time, they become cold, and sometimes more or less cyanotic. There is no impairment of sensibility; and no interference with the functions of the bladder, or, if at all, only for a few days. The functions of the rectum are commonly not interfered with, though obstinate constipation may exist.

All the characters mentioned in the last paragraph accord with those which present themselves in 'infantile paralysis,' but later on differences again show themselves.

One of the characteristic features in the child is arrest or rather diminished rate of growth in the parts affected, so that the limbs, or parts of limbs, paralysed remain, ever after, more or less abortive. This, of course, cannot occur in the adult; and also, owing to the fact that the joints are stronger, the secondary deformities due to secondary contractions taking place in certain non-paralysed antagonist muscles (often so serious in the child), are not met with to anything like the same extent in adults.

In regard to **diagnosis** the mode of origination of the disease; the fact that the paralysis is purely motor, and accompanied by no interference with sensibility; the fact that the reflexes are abolished, and that there is an entire absence of primary spasms; the fact that after the first few days, at least, the functions of the bladder and rectum are not interfered with; and also that in later stages there is atrophy of muscles, and the existence of the electrical 'reaction of degeneration'—these constitute a group of conditions which, taken as a whole, is thoroughly distinctive.

The disease with which it is most liable to be confounded is 'chronic spinal paralysis.' The points of distinction will be given under this head (p. 609). 'Progressive muscular atrophy,' if we bear in mind its very chronic onset, is much less liable to be confounded with the present disease, as also if we recollect that in it atrophy makes its appearance before paralysis rather than after, and that the electrical reactions are notably different (p. 621).

The fact of the absence of spasms, the diminution of reflexes, the non-interference with sensibility and with the sphincters, together with the abrupt origin of the disease, suffice, indeed, to separate the 'acute spinal paralysis' of adults from all other affections of the spinal cord.

For the first few days of its onset, if it induces a symmetrical paraplegic condition, and chances to be associated with paralysis of the bladder, an acute spinal paralysis may be confounded with an incomplete thrombotic 'softening' of the cord involving its anterior segment. The total disappearance of the reflexes should, however inspire doubt, and this doubt may be successively strengthened by a speedy resumption of power over the bladder, followed in about ten days by the development of the 'reaction of degeneration' and, subsequently, by the occurrence of atrophy in the paralysed muscles—the loss of the reflexes still remaining absolute. This is a combination of signs which could not occur from a focal lesion (*e.g.*, Softening) involving only the anterior half of the cord.

10. ACUTE ASCENDING PARALYSIS.

This is an obscure affection of the spinal cord, first definitely described by Landry in 1859 (and, therefore, sometimes named 'Landry's paralysis'), characterised on its clinical side by the existence of a progressive paralysis, advancing rapidly from below upwards, so as finally to implicate parts dependent for their innervation upon the medulla oblongata; characterised also on its anatomical side by the most puzzling absence of any appreciable pathological change.

On account of the latter peculiarity, the disease ought not to be referred to in the present place, but rather to be described among the functional diseases of the spinal cord (p. 592). But this disease, has such an amount of similarity with 'acute spinal paralysis' and 'chronic spinal paralysis,' from a clinical point of view, that it seems very desirable for their descriptions to follow one another, so that their mutual alliances as well as differences may be the more distinctly appreciated.

The disease seems principally to occur in persons between the ages of twenty and forty, and to be decidedly more frequent in males than in females. Although the brain and spinal cord of those who have died from this affection have now been frequently examined by skilled observers, the results have hitherto been entirely negative, so far as morbid anatomy is concerned.

All prodromata may be absent. When present there may, for a few days, or even for a few weeks, be a slight febrile condition from time to time, with a sense of weariness, and more or less numbness in the limbs, especially in the tips of the fingers and in the feet.

The disease then more definitely declares itself, by a marked weakness of the lower extremities; soon to be followed by actual paralysis, which, as in the 'subacute and chronic spinal paralysis,' shows itself first in the distal portions of the limbs, and gradually approaches the trunk, so that in the course of two or three days the paralysis of the lower extremities becomes complete.

The trunk muscles are next, and soon, implicated in a similar manner. The patient can no longer sit up or turn in bed. Respiration becomes more and more affected.

Soon, though sometimes after a distinct interval, the upper extremities become implicated; though here again the paralysis first involves the distal portions of the extremities, and thence gradually spreads (after a period in which mere paresis exists), till the whole limbs become completely powerless.

The paralysed limbs, both upper and lower, are lax, and show no trace of contraction. Though the muscles are flaccid, they do not undergo any marked amount of atrophy similar to that which occurs in the case of 'acute spinal paralysis.'

In accordance with this latter peculiarity, there is the further striking characteristic that the electrical reactions of nerves and muscles continue perfectly normal. This seems now to be a well attested fact, and it has been verified by good observers even after complete paralysis (without atrophy) has existed for several weeks.

Sensibility is scarcely, if at all affected; nor, as a rule, are pains complained of in the paralysed parts.

The nutrition of the skin is not impaired, so that there is no tendency to the formation of bed-sores. Coldness and a cyanotic condition of the limbs do not seem to be characteristics of this affection.

The sphincters are usually not at all affected. Constipation is often marked, and defæcation may be rendered difficult owing to paralysis of the abdominal muscles.

In regard to reflex actions, these—especially the skin-reflexes—may not be much affected at first, but may be abolished later on. Existing information is defective concerning 'tendon-reflexes' in this affection, and the writer has made no observations on the point himself.

As a rule there is no febrile elevation of temperature.

At the stage above indicated, in nearly one-third of the recorded cases, or it may be even before the arms have been much implicated, the disease becomes arrested, and, after a brief interval, recovery of power begins to manifest itself—usually in a reverse order, so that power is regained first over the arms, then over the trunk, and subsequently (in the course of several weeks) over the lower extremities.

But in the remaining two-thirds of the cases, after the arms have become paralysed, the disease still progresses so as to affect the cervical muscles, the diaphragm, and finally the muscles innervated by the motor nerves of the medulla. Thus, in its later phases the disease is characterised by a greatly increasing difficulty in respiration; great weakness in voice; extreme rapidity of pulse; and possibly by inequality of the pupils. Finally, increasing paralysis of the muscles concerned with articulation and deglutition sets in; and, owing to the augmenting difficulties of respiration, death may arrive at any moment by asphyxia.

This climax of the disease may be reached in the course even of three or four days; on the other hand, it may not be reached until as many weeks have elapsed. Whenever the disease has advanced so far as seriously to implicate the medulla, recoveries are comparatively rare.

In quite exceptional cases, the disease may pursue a reverse order throughout; implicating the nerves of the medulla first, then those of the cervical region of the cord, and so on. The celebrated Cuvier is said to have died from the disease, progressing in this very unusual manner.

From the point of view of **diagnosis**, it is clear that, so far as the established disease is concerned, we have in this affection, in 'acute spinal paralysis of adults,' and in the sub-acute forms of 'chronic spinal paralysis' maladies that present certain well-marked points of similarity. In each we have to do with simple motor paralysis, with no fever, no tenderness or pains in the spine, no pains in the limbs or contractions, and with no incontinence of urine or fæces, or tendency to the occurrence of bed-sores.

'Acute ascending paralysis' differs from both these affections, however, in the important fact that rapid atrophy does not set in in the paralysed muscles, and that the electrical reactions in no way differ from those met with in healthy nerves and muscles.

In the very acute cases, of a few days' duration only, these distinctions would be worthless, as sufficient time would not have elapsed to make it possible for either of these signs to occur. In such rapid cases, therefore, the distinctly progressive character of the disease is that which will serve to distinguish it from the more severe cases of 'acute spinal paralysis,' in which the paralysis sets in simultaneously throughout the whole of the parts affected, and often with a pretty distinct initial febrile disturbance. Then, again, there is the fact that this latter disease has no tendency to involve the medulla, and is only very rarely fatal.

It is in the diagnosis of the more slowly evolved forms of 'acute ascending paralysis,' from the similarly progressive cases of 'chronic spinal paralysis,' that the development of rapid atrophy of the muscles, together with the 'reaction of degeneration' comes to be distinctive of the latter affection. Then, again, in 'acute ascending paralysis,' there is a longer persistence of reflex actions, and a far greater tendency to the manifestation of symptoms showing that the medulla oblongata is involved.

We must now give some details concerning five forms of paralysis, also apt to be acute in their mode of onset, though they are all more or less rare forms of paralysis of spinal origin, and none of them are known to be associated with definite pathological changes—they belong, therefore, to the category of so-called **Functional Diseases of the Spinal Cord.**

11. TOXIC SPINAL PARALYSES.

Under this name reference must be made to a class of cases of paraplegia produced by poisons of various kinds. It constitutes a somewhat heterogeneous group, concerning which our knowledge is still very defective—in the main, because such cases are of rare occurrence.

Of the toxic agents taken into the body, and capable of entailing a more or less complete paraplegic condition, some are minerals, such as arsenic and lead; others are of vegetable origin, such as aconitia, conia, veratria, prussic acid, ergot, and alcohol; while others again are of animal origin. In the majority of cases, their action as 'causes' is not sufficiently potent to lead to paralysis with anything like uniformity. They need the concurrence of other favouring circumstances, probably in the main intrinsic; but, under the combination of conditions thus resulting, a paraplegia may be induced—in actual modes, however, that may differ considerably among themselves in different cases. It is only in this attenuated sense that the above-mentioned poisons are to be regarded as 'causes' of paraplegia. They ought perhaps, from this point of view, to be considered as predisposing rather than as exciting, and in no case as proximate, causes of paraplegia.

This holds good, for all that we know, concerning the fitful and irregular manner in which arsenic, lead, and alcohol (and probably, to a similar extent, other toxic substances) give rise to paraplegic symptoms in those who have taken them to excess.

Thus, in regard to **arsenic,** it is true that in certain cases Orfila observed paraplegic conditions in dogs which had taken large quantities of this drug; but such symptoms would seem to be met with only occasionally as a result of acute arsenical poisoning in man, and, perhaps, with equal rarity in those who have habitually consumed large quantities of this substance.

Again, according to Tanquerel des Planches, out of 200 cases of **lead-poisoning,** in only fifteen did the paralysis produced implicate the lower extremities; and in one only of these did it occur as a distinct paraplegia. This case might well, therefore, have been due to some accidental combination of conditions—in short, it might have been a coincidence rather than a definite result of the taking of lead; or if, in any sense, a result of this antecedent, then a result of it only in part and in conjunction with other co-operating causes.

Lead-poisoning may act in more ways than one in bringing about paralysis. It commonly induces a condition of lowered vitality, which may favour the development, in different cases, of this or that degenerative condition of the spinal cord. In such cases, it can generally only be considered as one among other determining conditions, tending to bring about one or other form of paralysis. It has seemed, for instance, to act as one factor in the production of 'spasmodic paralysis' on one or two occasions; more frequently, according to Erb, it has seemed to have some share in the production of a 'chronic myelitis'; while, according to Bramwell, in certain cases it favours or may seem to be the actual cause of progressive muscular atrophy. A case of this latter type has recently been under my care at University College Hospital.

The more specific effects of lead as a poison are, however, seen in the production of a well known form of paralysis, which principally affects the extensor muscles of the upper extremities, causing them to atrophy, and leading to the well-known condition commonly characterized as 'wrist-drop'—the electrical reactions of the atrophied muscles being those comprised under the 'reaction of degeneration' (p. 10).

In regard to this specific form of paralysis, it has been considered doubtful whether to regard it as one of a peripheral type, or as a paralysis due to some change in the spinal cord. The evidence seems, however, decidedly to incline towards the latter view. On this point Erb says:—"In speaking of the pathogenesis of lead paralysis, which is such a peculiarly characteristic affection, I have sought to show that its origin is probably spinal instead of peripheral, and E. Remak has established this more accurately and in a more detailed manner by the sifting and critical examination of a larger amount of material. He comes to the conclusion that quite circumscribed alterations in the grey anterior horns probably lie at the foundation of lead paralysis. These alterations might well be of a degenerative or chronic inflammatory nature, but are, as a rule, capable of resolution. Bernhardt has, also, recently given in his adherence to this view. In fact, the whole state of things in lead paralysis, with reference to mobility, atrophy, electrical relations, the absence of disturbances of sensibility, etc., is so entirely analagous to that in poliomyelitis anterior chronica, that we are almost forced to the theory of changes in the anterior grey horns in lead paralysis. On the other hand, again, I recently saw two cases of paralysis of the upper extremities, which, in all their details, were so completely analagous to lead paralysis that only the entire lack of any sort of evidence of lead-poisoning was sufficient to determine me to give up this diagnosis, and accept that of a chronic circumscribed poliomyelitis anterior."

In confirmation of these predictions it should be stated that Oppenheimer has recently recorded a case of severe lead paralysis with autopsy ('Lancet,' Nov. 14, 1885) in which a sclerosis of the anterior horns was found, principally in the cervical and in the lumbar regions of the cord, associated with a complete atrophy of the great ganglion cells in these situations.

Probably the mode of action of **alcohol** is even more general and ill-defined. Wilks, however, goes so far as to speak of an 'alcoholic paraplegia,' resulting from excesses in spirit drinking. Alcohol like many poisons, when taken in undue quantity, deteriorates the nutrition of the body generally ; it may spoil the integrity of its more delicate tissues, and thus interfere with the discharge of many different functions. In this way, through general spoiling and degeneration, the way may be led on to the development of special changes in the nervous system favouring the occurrence of paralysis in one or other part of the body. If either one of a group of possible morbid changes, induced upon such a basis, chances to affect principally the lower part of the spinal cord (which seems a part specially prone to be implicated in such cases) a paraplegia may be induced. The principal justification, however, for speaking of such a state as an 'alcoholic paraplegia,' probably lies in the fact that here (as, indeed, in all cases of toxic paralysis) the first therapeutic indication is to be found in the renunciation of the harmful agent.

According to Wilks, 'alcoholic paraplegia' is more frequently met with in women than in men, and is brought about especially by the habit of constantly drinking brandy or other spirit to excess. He says :—" Drunkards of all descriptions suffer from muscular and nervous weakness, but, as I before said, it is more especially in the legs that the effect is most striking. A loss of power is first observed, accompanied by pains in the limbs, and in some cases by anæsthesia, which seems to indicate a chronic meningitis of the spinal cord. There is, at the same time, necessarily some amount of feebleness of other parts of the body as well as of the mind, and thus an approach to general paralysis is produced ; but sometimes the symptoms are almost confined to the legs and resemble those of locomotor ataxy. Why the brain should be affected in one case, and the spinal cord in another, is due probably to the same idiosyncrasy which makes one man get drunk in the head and another in the legs. . . . . I might add that drunkards often suffer from pains in the limbs long before there is any sign of paralysis " ('Diseases of the Nervous System,' p. 224).

Dr. Broadbent has described a form of 'Alcoholic Paralysis' in which the upper as well as the lower extremities are affected, to which he assigns the following characters ('Med. Chirurg. Trans.,' 1884, p. 141).

" There is first gradually increasing weakness of the lower extremities which may be noted for some time, when marked loss of power becomes manifest in the extensor muscles of the forearm, giving rise to double drop-wrist. It cannot be said that when this is first apparent there is no distinct paralysis elsewhere ; the lower extremities and the back are weak, but the drop-wrist may be complete, and the flexors of the hand may also be paralysed so that the hand is like a flail, while the movements of the elbow and shoulder are good and fairly vigorous, and the patient can support himself on his feet. As the paralysis advances, all the muscles of both upper and lower extremities are affected, and the limbs can only just be moved, the arms being, as a rule, more seriously implicated than the legs; the muscles of the trunk also are paralysed so that the patient cannot raise himself or turn in bed. The sphincters retain their functional power, and sensation is not affected. There is no pain from first to last, though the muscles may be tender on handling." In one exceptional case, however, Broadbent says, "sharp pains shot down the legs from time to time, and there was incontinence of urine." There is no fever and no spasm. A remarkable loss of tone in the capillaries exists, so that when the patient is seated on the edge of the bed with the legs hanging down, the feet become filled with blood and can literally be seen to swell and become puffy. It is the degree of capillary paralysis which is remarkable, and the early period at which it is observed. The knee-jerks are absent. There are no signs of implication of the medulla. In cases which prove fatal, "Death is caused eventually by paralysis of the respiratory muscles, and the diaphragm is the first of these to be affected. . . . Later, the muscles of the chest become affected, apparently from below upwards, when, of course, death ensues from apnœa." In one case, examined post-mortem, Broadbent was, after careful examination, unable to discover either macroscopic or microscopic changes in the spinal cord. He thinks the affection is most closely related to 'acute ascending paralysis' and does not hesitate to regard it as a spinal malady. In regard to causation, he says, "It would appear that unrestricted access to stimulants throughout the entire day, and an indoor inactive mode of life, were the conditions which together invited the disease; these were just the conditions common to the five women and two men who have come under my observation."

It is unfortunate that neither of these observers has given the electrical reactions of the paralysed muscles in the cases they have recorded, and without exact information on this point, in regard to both induced and constant currents (whether for instance the electrical 'reaction of degeneration' exists or not), it is absolutely impossible to discuss, with any advantage, the alliances of the affection described by Broadbent. It is equally clear that his form of alcoholic paralysis is quite different from that described by Wilks. It is worthy of remark also, in this connection, that Dr. Hadden has since recorded two other cases of so-called 'alcoholic paralysis' ('Path. Trans.,' 1885, p. 54) the clinical characters of which do not accord with either of the types above referred to, and that he, moreover, supports a view of the pathology of alcoholic paralysis which is quite different from that supported by Broadbent, or assumed to exist by Wilks (the latter supposes a chronic meningitis or meningo-myelitis) since he says, "although our knowledge of the pathology of alcoholic paralysis is as yet very scanty, there seems to be little doubt that the lesion is essentially one of the peripheral nerves. According to Lanceraux and Dreschfield the alterations begin in the nerve fibres themselves, but in the second of my cases it had rather the characters of a peri-neuritis or sclerosis."

All these divergences of result and opinion, decidedly strengthen me in my notion that the name 'alcoholic paralysis' is an unfortunate one; that at present there is no one condition which answers to this name; and that its adoption will tend to encourage the making of an easily arrived at, slip-shod diagnosis, or what seems to be such, when in reality no diagnosis in the proper sense of the term has been made. There is great danger, in fact, that the term 'alcoholic paralysis' may soon come to be just as meaningless, and, it may be added, erroneous, as its compeer 'hysterical paralysis' is too often found to be.

The notion has recently been advanced by Moxon, that a certain class of poisons which own the common property of being 'depressants of the circulation,' have also a tendency to paralyse the hind legs rather than the fore legs of animals. In this group of poisons are included **aconitia, conia,** and (though doubtfully) **veratria**; as well as **chloral** and **prussic acid.** He thinks these drugs act by causing further impediments "to the exceedingly and peculiarly difficult blood-supply of the caudal end of the spinal cord" ('Brit. Med. Journ.,' April 2nd, 1881, p. 498). In reference to this view, it should be borne in mind that extreme feebleness of blood-current is of itself a common cause predisposing to the occurrence of thrombosis both in arteries and veins, and that such a condition may intervene in some of these cases of poisoning, and thus lead to the development of the paraplegia. This would enable us to account for the otherwise inexplicable fact of the maintenance of the paralysis long after other effects of the poison have passed away—as seems to be not unfrequently the case. This being so, any such paralysis could only be counted as a toxic paralysis, in a very indirect fashion. Nothing more definite can, I believe, be said in regard to the possible paralysing action of this class of poisons.

12. INTERMITTENT PARAPLEGIA.

Very few cases of paraplegia of this type have been recorded, and it must certainly be a condition of extreme rarity.

The earliest recorded example was made known by Romberg, and as this, both in its nature and its course, seems to have been a typical instance, it may be cited here. 'A woman, sixty-four years of age, after being quite well the day before, was suddenly attacked with paralysis of the lower extremities and of the sphincters. Sensibility was unchanged, consciousness clear, the temperature cool, pulse 80, small and empty, no pain in the spinal cord. The next day there was an astonishing change in the condition. The patient could walk again and void urine voluntarily, and only complained of weakness in the legs. The following morning there was paraplegia again, which had set in at the same hour as it had done two days before. A third paroxysm was awaited, which also set in at the appointed time, although without paralysis of the sphincters. Quinine effected a rapid cure.'

Two other cases are cited by Erb. In one of them, there were also three attacks before cure took place under the influence of quinine; but in the other, observed by Hartwig, attacks of intermittent paralysis seem to have gone on for many months. It is worthy of note that, in the two former cases, there is no statement that the patient had previously suffered from ague; while, in that of Hartwig, although the man had been afflicted with tertian intermittent fever five years previously for a few weeks, there is no mention made of the recurrence of any other symptoms of this type, even during the period that the patient continued subject to the attacks of intermittent paraplegia.

We know absolutely nothing as to the real cause or intimate pathology of such attacks. Any future cases, therefore, deserve to be observed and recorded with the greatest care. Meanwhile, it should be remembered that the cases already observed seem to have proved extremely amenable to the influence of quinine and of arsenic.

13. HYSTERICAL PARAPLEGIA.

Great difference of opinion exists as to the frequency of any such affection. The same remark, however, may be made concerning hysterical paralyses generally. Some think they see them frequently; others regard such affections as rarities.

It is admitted by all, that paralyses of an hysterical type more frequently belong to the paraplegic than to the hemiplegic variety.

Hysterical paraplegia is commonly incomplete, and the limbs affected may be either flaccid or rigid. Definite characters are, however, wanting throughout. Reliance is always placed principally upon the absence of distinct evidence of organic disease, such as would be furnished by muscular atrophy together with the electrical 'reaction of degeneration,' by incontinence of urine or of fæces, and by the occurrence of bed-sores.

Beyond these negative characters it is most important to look to the previous history of the patient, to the course of the disease, and for the existence (or for the history) of sudden variations in important symptoms. The coexistence of other evidences of hysteria is of little absolute value, because it is so common to find symptoms of a so-called hysterical order associated with real organic disease either of the spinal cord or of the brain.

The electrical reactions are usually normal, though there may be a slightly lowered irritability to both currents. Variations in sensibility are common. Thus, the muscles may be so insensitive that the strongest faradic currents produce no pain. There may also be analgesia of the skin in some regions, in which sensibility to touch and temperature are preserved. In other skin regions there may be marked hyperæsthesia. These anomalies in regard to sensibility are apt to be temporary and shifting in their seat. Variations in degree of motor power may also be met with from time to time.

In such cases, there may also be marked exaggeration of the deep reflexes, that is the existence of ankle clonus on one or both sides as well as an unduly free knee-jerk. It is especially important to bear in mind that marked ankle clonus may be present, independently of actual organic disease. Similarly, no absolute weight can be attached to the absence of knee-jerk, as deciding against a functional malady and in favour of organic disease. Too much importance has been attached to this sign. The writer has seen many cases—partly children, partly young women, and partly adults—in which although there were no symptoms pointing to spinal disease, no knee-jerk could be elicited under the most favourable conditions, that is, with bare knee and use of percussion hammer. In many of the cases, these results have been confirmed by several examinations at different times. They seemed to be individual peculiarities, and in no way indicative of cerebral or of spinal disease.

As I have said before, therefore, the diagnosis of hysterical paraplegia will rarely, if ever, be legitimately made upon the mere positive characters presented by the patient. The cautious and skilled practitioner will be inclined to place much more weight upon the absence of certain signs or peculiarities, the presence of which is clearly indicative of organic disease of the spinal cord.

In short, the real question that presents itself in all these doubtful cases is—have we, in the case before us, to do with a functional disorder or with an organic disease in the spinal cord? Some even good observers are rather too prone to look upon the terms 'functional' and 'hysterical' as interchangeable. This the writer considers a regrettable error, tending to create confusion. Surely there may be functional paralyses which have no right, merely as such, to the appelation 'hysterical.'

Having satisfied ourselves that the disease is not likely to be organic, we, upon this basis only, may assume that we probably have to do with a functional disorder. We gain nothing by calling such a case hysterical. There is oftentimes no warrant for so doing. The use of the term may, and does, seem to imply some extra definiteness of knowledge on the part of him who uses it; and yet a close examination of his position, and of his grounds for employing the term, would probably soon convince his questioner, if not himself, that he does not possess any superior definiteness of knowledge—and that he is only in a position to say that the probabilities are more or less strongly in favour of the view that the patient is suffering from a functional disorder, rather than from an organic disease, of the spinal cord.

In a certain proportion of these cases, the patient presents no other symptoms or mental peculiarities that could be described as hysterical. By myself, therefore, such a term would never be applied to one of these cases—I should simply speak of it as a case of functional disorder.

In other instances, it is true, a paralysis may coexist with distinct hysterical symptoms. These are, however, precisely the cases in which the greatest amount of care and circumspection are needed before coming to the conclusion that the particular case before us is one of hysterical paralysis. I have no hesitation in saying that cases of hysterical paraplegia, rightly so called, are only encountered with extreme rarity; and that, on the other hand, cases of organic disease of the spinal cord, associated with more or less marked hysterical symptoms, are not uncommonly met with—and that, moreover, a by no means inconsiderable percentage of these cases are regarded by this or that practitioner as cases of 'hysterical paralysis.' Such, at least, are the cases that come under my own observation not unfrequently.

In this latter class of cases, it is perfectly true that the symptoms are often largely in excess of those due to the actual organic disease. We have symptoms of a more permanent and incurable type due to the organic disease, and beyond these a pretty broad fringe or margin of symptoms of a curable and changeable order, due to functional exaggeration through the influence of fear, fancy, or other less definable mode of causation. The recognition of this may enable us confidently to predict a speedy mitigation of symptoms as possible in such cases.

'Disseminated sclerosis' in its early stages is a disease which is peculiarly apt to be confounded with hysterical paralysis. This disease is not at all commonly known to the great majority of practitioners, and is often difficult of diagnosis in its early stages. Not being able to identify its manifestations with those of any organic disease that is well known to them, observers of it are often apt, when they meet with it, to regard it as functional and to call it 'hysterical.' This is, in part, all the more pardonable, because even those who have the largest experience of such diseases as 'disseminated sclerosis,' at times meet with cases, which, in their early stages, can only be separated with the greatest difficulty from such functional disorders as are rightly termed hysterical. The difficulty of diagnosis in some of these cases is, in fact, extreme.

A similar difficulty is also, at times, apt to present itself in the recognition of 'primary lateral sclerosis' (spasmodic spinal paralysis), when it occurs in young women, and with a similar result so far as diagnosis is concerned.

The other organic diseases which are, perhaps, most frequently apt to be confounded with hysteria when occurring in young women are 'subacute' and 'chronic spinal paralysis'; and also, the early stages of the paralysis associated with 'Pott's disease,' in cases where the angular curvature is slight and undetected. But in both sets of cases, only ordinary care is needed to prevent any such errors being made.

In conclusion, I would, therefore, call the student's and practitioner's attention to the following points, in connection with the subject of this section :—

Where he has only reason to believe that in a given case he has to do with functional paralysis, he should not necessarily and in the absence of other good reasons, speak of it as 'hysterical paralysis.'

That 'hysterical paralysis' is really an extremely rare affection, and is a diagnosis most frequently arrived at by those who are somewhat defectively equipped in regard to any special knowledge of diseases of the spinal cord.

One cause, therefore, why the suspicion of the existence of such a malady is much more frequent than its actuality, is that mistakes in diagnosis are frequent, and that the early, or sometimes even the late, stages of various organic diseases are erroneously labelled as cases of 'hysterical paralysis.'

Another reason why such a diagnosis is made much more frequently than the actual facts would warrant, is to be found in the fact that various organic diseases of the spinal cord, especially in young women, are apt to be associated with well-marked hysterical symptoms. This fact being ignored, or too much weight being given to the hysterical symptoms, a hasty diagnosis is apt to be made, and the case is termed one of 'hysterical paralysis,' when, at the most, the symptoms are only aggravated by coexisting hysteria, and the real cause of the malady lies in some unrecognized form of organic disease.

Finally, it should be distinctly borne in mind that neither functional paralysis nor its more special form 'hysterical paralysis' can ever be safely diagnosed except by way of exclusion. We must first, by the most careful examination, thoroughly satisfy ourselves that we have not to do with either of the many forms of organic disease of the spinal cord or its membranes, before we can, with any safety, come to the diagnosis that the case before us is one of functional or of hysterical paralysis.

The result of what has been said goes to show that the diagnosis of 'hysterical paralysis' is one which should never be lightly made, since to make it securely is one of the most difficult problems—demanding, as it does, on the part of the practitioner a preliminary confident opinion that the symptoms in the case before him are not explicable by any known organic disease of the spinal cord.

14. PARAPLEGIA DEPENDENT ON IDEA.

This is a form of paralysis, of purely 'functional' type, occasionally occurring in neurotic impressionable persons. Notwithstanding its usually slower mode of onset, it seems best to refer now to this variety of functional paralysis.

Attention was first called to this class of cases by Dr. Russell Reynolds, who cited, amongst others, a typical instance in which a young lady, while attending to a paraplegic father, amid the additional anxieties consequent upon straitened circumstances and the fatigues incident to teaching in order to obtain the bare necessaries of life, at last, under the influence of long-continued strain, together with an abiding fear (inspired by actual physical weakness) that she herself was becoming paralysed, became reduced *de facto* to this condition, as the final outcome of a slowly-increasing feebleness (see 'Brit. Med. Jrnl.,' Nov. 6, 1869).

Such a condition may occur quite independently of hysteria, and be just as free from anything like conscious simulation or desire to exaggerate. We cannot say positively that the state is induced by what is called 'inhibition,' or by definite vascular spasms such as are supposed to form one of the pathological bases of the class of so-called 'reflex' paralyses, and yet both these modifying influences over the functional activity of the spinal cord *may* be in part operative when imagination, continuously excited in some one direction, has a tendency to pervert the functional activity of this portion of the nervous system.

The same conditions that exist as more lasting states in these cases, probably exist temporarily, under the influence of suggestion, in hypnotised persons.

In regard to the symptoms met with, there is a paralysis of motion in the lower extremities, more or less complete, often partial, and generally without implication of sensibility. There is unabated control over the bladder and rectum.

Dr. Reynolds points out that, while such patients may be apparently quite incapable of lifting a foot from the bed, they often find themselves able to turn or sit up without any assistance. And, in slighter cases, though they may be unable to stand for a moment, such patients may yet be able to move the legs in any direction while in the recumbent position.

From the point of view of **diagnosis** the character of the paralysis, and its limitation in range, is thought to be of importance. But still more important is the establishment of the fact of the pre-existence of long-continued fears or fancies (in a person of delicate or neurotic temperament) of such a nature as to be in accordance with the patient's now present condition, combined with the absence of all signs positively indicative of any structural defect in the spinal cord.

Where such a condition presents itself (as it may) as a mere complication of an actually existing structural disease, the diagnosis becomes either impossible or extremely difficult. It is, in fact, only possible after prolonged observation and experience as to the course of the symptoms.

15. REFLEX PARAPLEGIA.

Some practitioners believe that paralyses of various kinds are brought about purely by reflex influences. They would include under this category some of the cases of paralysis of separate muscles, such as the ocular; some cases of paralysis of one or both arms; or some of the cases of paralysis of one or both lower extremities. It is the latter class of cases with which we are now specially concerned, though much of what is to be said under this head may, *mutatis mutandis,* be considered applicable to the whole class of so-called 'reflex paralyses.'

Those who believe in the common occurrence of this form of paralysis are considerably less numerous than they were five-and-twenty years ago, when the notion of its frequency and importance was warmly espoused by Brown-Séquard ('Lects. on Paral. of Lower Extremities,' 1861), and when the morbid anatomy of the spinal cord was still very imperfectly known. The number of competent observers at this time was smaller, while the difficulty in detecting morbid changes in the spinal cord was much greater then than it is now that we are accustomed to employ more elaborate methods for its preservation as well as for its examination. Yet one of the strongest of the arguments brought forward in favour of the existence of 'reflex paraplegia' was the absence of discovered lesions in the spinal cord in a class of cases reported upon by Stanley in 1833 ('Med.-Chir. Trans.,' Vol. xviii., p. 260) in which paraplegia was associated with various morbid conditions of the urinary organs—cases, in fact, of the so-called 'urinary paraplegia.' And the main support for the opinions of those who still believe in the existence of a class of reflex paraplegias, now, also, lies in the absence, in certain cases of paraplegia terminating fatally, of any actually-discovered lesion.

The interpretation or pathogenesis of the paralyses of this class put forward by Brown-Séquard is as follows:—That an irritation, operating upon certain sensory nerves, produces impressions which, after impinging upon the properly related grey matter in the spinal cord, are thence, in part, reflected along vaso-motor nerves regulating the calibre of certain blood-vessels which supply either (*a*) the portion of the spinal cord in relation with the paralysed parts, or else (*b*) the great nerves or the muscles themselves of the paralysed parts. In either case this reflection of impressions resulting from irritation of sensory nerves, from related portions of the grey matter of the spinal cord, upon such special groups of vaso-motor nerves, is supposed to lead to a persistent spasm of the vessels which these nerves innervate, so as to cause a continuous anæmic condition, either of certain vascular territories in the spinal cord itself, or else of the related nerve-trunks and muscles. In either case, too, the nutrition of the parts involved in this anæmia is supposed to suffer—so that their functions can no longer be carried on, or only in a very imperfect manner—and thus a more or less complete paralysis is thought to result—capable, however, of being mitigated from time to time, of actually intermitting, or indeed of being abruptly cured, according as temporary diminutions or a complete disappearance of the original exciting cause, may lead to a diminution or to an actual cessation of the supposed profound anæmia produced by the supposed spasms of vessels. These are the theories and suppositions upon which the doctrines of 'reflex paraplegia' are based.

Among the sources from which the initial irritation is supposed to proceed, almost all parts of the body, internal as well as external, are cited. Thus, irritative impressions, it is thought, may emanate from almost any part of the urinary tract—from the urethra to the kidney; in other cases, similar impressions may emanate from some portion of the female genital organs; in others, from the intestinal canal, owing to the presence of worms or some such persistent causes of irritation; in others, from some portion of the thoracic organs; or, as it seems to be thought, from irritated sensitive nerves in almost any part of the body, whether situated near the surface or deep among the tissues.

The assemblage of symptoms supposed to characterise these forms of reflex paralysis presents nothing like a distinctive mode of grouping. And of the several components of the group put forward by Brown-Séquard in 1861 (*loc. cit.*, p. 33), as pertaining to one of the most typical varieties, namely 'urinary paraplegia,' none can have any pretensions to be regarded as distinctive, excepting the alleged tendency of the paralysis to vary in degree with variations in the malady on which it is supposed to depend, together with its tendency to spontaneous or easy cure coincidentally with or soon after the cessation of the urinary troubles, whatever they may have been. In harmony with this latter character, also, are the alleged facts that speedy cures have been brought about of cases of paraplegia, especially in children, after the expulsion from the alimentary canal of tape-worms or round-worms; or, of cures of the same disease in adult females after the cessation of some uterine inflammation; or of cures of a paralysis of ocular muscles after the removal of some carious tooth, which had previously been exercising an irritative influence upon branches of the fifth nerve.

It would be improper and useless to deny the existence of such cases; they are theoretically possible. On the other hand, the writer is compelled to believe, after a very extensive experience, that, if they exist, they can only occur as extremely rare events.

Although it is theoretically possible that an irritation of a sensory nerve may be reflected on vaso-motor nerves, so as to lead to arterial spasms in certain territories of the spinal cord or in certain groups of muscles, no proof exists in favour of the view that such a condition of spasm could be maintained for weeks or even months. Nor, if it could occur for these prolonged periods, and to such an extent as to annul some of the most important functions of the spinal cord during this time, is it at all clear that the nutrition of the cord in the affected regions would not be seriously interfered with by such prolonged anæmia; and if so, the assumed speedy resumption of healthy functions after the disappearance of the vascular spasms would constitute another difficulty, since such speedy recovery would be incompatible with the theory upon which the explanation of the disease is based.

Again, it is almost certain that many of the cases formerly supposed to belong to this category of 'reflex paralysis' had no right to figure therein. Cases of 'diphtheritic paralysis' have been proved to belong to a different class; and there is good reason to believe that in other instances the morbid conditions really existing as causes of the paralysis have simply been overlooked, either because the appreciable changes were only slightly advanced at the time of the patient's death, owing to the brief duration of the illness; or because of the want of a thorough examination of the cord, conducted with all needful aids, care, and expenditure of time.

It seems clear, therefore, that the opinions of those who believe in the existence of 'reflex paralysis,' and of 'reflex paraplegia' in particular, stand much in need of further support. Well-observed and well-recorded instances of the disease are urgently wanted, if reflex paraplegia is to retain its claim to a place in our nosology.

SPINAL PARALYSES OF SUB-ACUTE ONSET.

Under this head we have paralysis resulting from the following causes :—

16. Sub-acute and Chronic Spinal Paralysis.

17. Scrofulous Pachymeningitis, associated with Vertebral Caries.

It should be borne in mind that some of the forms of paralysis already described, viz., Nos. 10-15 *inclusive, may have a Sub-Acute Onset, and, on the other hand, that No.* 17 *may have a Chronic Onset (see p.* 562*).*

16. SUB-ACUTE AND CHRONIC SPINAL PARALYSIS.

This disease was believed by Duchenne, who first described it, to be dependent upon a chronic degeneration occurring in the grey anterior horns, and this view is supported by the few examinations as yet made of persons who have been the subjects of this affection. The pathological changes in the anterior horns have been associated with atrophy of the anterior nerve-roots.

The causes of the malady are at present almost wholly unknown ; but it occurs principally in individuals between the ages of thirty and fifty years. As with other chronic spinal affections, so with this, there has often been one or other of the following events occurring some little time before the onset of the disease :—Exposure to cold and damp, some shock or concussion, venereal excesses, or great fatigue induced by other causes. But what share the pre-existence of one or other of these conditions may have had in initiating the disease cannot at present be defined.

In the sub-acute cases, paralysis may become developed (usually in the lower extremities first) in the course of a few days or weeks; at the same time, there may be some very slight initial febrile disturbance, and possibly some shooting pains in the back and limbs.

In the more chronic cases, the latter symptoms may be absent, and the onset of paralysis is very much slower. There may be, at first, mere paresis, felt most in the ankles and knees ; but gradually (often after many months) this deepens into distinct paralysis of certain groups of muscles, or of the entire limbs. The muscles are flabby and progressively waste ; at the same time they cease to respond well, or even at all, to the faradic current, and become more sensitive to the voltaic. There may, also, be notable fibrillar twitchings in the muscles undergoing this atrophic process.

Sensibility is unaffected. Skin and tendon reflexes in the paralysed regions are abolished. The temperature of the affected limbs is lowered; and the feet especially are apt to be cold and cyanotic.

Soon the arms become affected in a similar manner, and here the paralysis may first affect either the extensors or the flexors. It may remain more or less limited to certain groups of muscles, or may gradually extend so as to implicate the whole limb. The distal parts are usually, however, more completely involved than the proximal. In the arms the same kind of phenomena occur as in the lower extremities, and there is a similar absence of rigidities or contractures.

There is no tendency to the formation of bedsores, and the nutrition of the skin seems to be unimpaired.

The rectum, the bladder, and the sexual organs are usually quite unaffected.

After a time, the excessive reaction of the wasted muscles to the galvanic current decidedly diminishes; though in the earlier stages of this affection the electrical 'reaction of degeneration' exists with all its characteristic details (see p. 10).

In the subacute cases, after a month or two, improvement may gradually begin to manifest itself; and, in exceptional instances, this may go on slowly, but steadily, to complete recovery. In other of these cases, however, certain muscles or groups of muscles do not undergo the same improvement as the others; they may continue paralysed, and become more and more atrophied.

In the more chronic cases, recovery is less likely to occur; though after the symptoms have developed to a certain extent, it occasionally happens that no further advance is made. Such patients may remain in much the same condition for years.

In another class of cases, the malady proves more continuously progressive. After implicating the upper and lower extremities severely, the morbid process may extend to the upper cervical region of the cord, so as greatly to interfere with respiration; or it may even extend to the medulla, so as to involve the tongue and pharyngeal muscles, and more or less interfere with the functions of articulation and deglutition. In such cases death is liable to occur through asphyxia or slowly progressing exhaustion.

In not a few cases of this disease, more or less complete recovery occurs, though it may be only after from two to four years.

From the point of view of **diagnosis** this malady bears a closer resemblance to the 'acute spinal paralysis' of adults than to any other affection. The two diseases are naturally distinct in their modes of initiation, but as established diseases (that is, in their later phases), they would be very difficult to discriminate from one another, in the absence of definite information as to modes of onset—and such information is sometimes not obtainable. It is the abrupt commencement of the paralysis over a wide area of the body that is met with in, and which is so distinctive of, 'acute spinal paralysis'; while in the 'sub-acute' forms, and more especially in 'chronic spinal paralysis,' we have to do with a distinctly progressive spread of the disease from part to part.

In regard to the discrimination of these 'sub-acute and chronic forms of spinal paralysis' from some other varieties of spinal cord disease, the reader may refer to what has been said concerning the grounds on which the diagnosis of 'acute spinal paralysis' is to be made (p. 588).

In 'amyotrophic lateral sclerosis' the upper extremities may be paralysed, wasted, and flaccid as they are in 'chronic spinal paralysis'; but then in the former disease there would be the characteristically different combination of paralysis, without wasting, but with more or less rigidity, in the lower extremities.

For the distinguishing characters of 'acute ascending paralysis' see the account of that affection (p. 589).

17. SCROFULOUS PACHYMENINGITIS, ASSOCIATED WITH VERTEBRAL CARIES.

One of the most frequent causes of 'Slow Compression of the Spinal Cord' is to be found in diseases of the vertebræ, and especially simple inflammatory or scrofulous caries of the bodies of the vertebræ (leading to *angular curvature*, or 'Pott's disease'). Still, other kinds of disease of the vertebræ may also be productive of slow compression of the spinal cord, and of that form of localised softening of the organ which is so commonly met with in this class of cases (the so-called 'compression myelitis'). Among these may be mentioned *cancer* of the vertebræ, either primary or secondary; also *exostoses* projecting into the spinal canal, or more irregular thickening of the bones in this situation, such as are met with occasionally in *syphilitic hyperostosis* or in *rheumatoid arthritis* (see Table II., p. 561).

In cases of vertebral caries, a tough, yellow, scrofulous growth often infiltrates the posterior vertebral ligament, and thence spreads to the dura mater, here producing thickening and irregular fungosities which may press injuriously upon the spinal cord—more especially upon its antero-lateral columns. In these cases, the organ may be distinctly softened opposite, and perhaps for a very short distance above and below, the site of compression. At first such softening is principally apparent in the columns above mentioned; but in cases of longer duration it may involve the whole thickness of the cord, and be followed by the usual ascending and descending 'secondary degenerations.' The softened matter itself is an almost bloodless fluid or semi-fluid pulp, either of a whitish or dull yellowish-white colour; and there is generally no undue vascularity of the immediately adjacent portions of the cord.

In certain other cases of slow compression no such softening of the cord is produced; there is rather a slow atrophy or disappearance of the nerve-substance as the pressure increases, together with a sclerosis of what remains. This is most apt to occur, however, where the cord is pressed upon by some exostosis, or by irregular growth and thickening of the inner surface of the spinal canal, such as takes place occasionally in one or other of the cervical vertebræ.

It has long been known that no constant relation exists between the amount of angular curvature and of paralysis in different cases of vertebral caries. Paralysis may be absent where curvature is most marked. On the other hand, with no curvature and with only a slightly marked projection of one or two vertebral spines, paralysis may yet exist to a well-marked degree. This is due to the fact that in such cases the cord is only very rarely compressed by the bones, while it is frequently more or less pressed upon by the yellowish growths which protrude from the inflamed or carious vertebræ, or which produce thickening and infiltration of the dura mater at the seat of disease. Changes of this sort may be well-marked even where no angular curvature is appreciable.

Again, where angular curvature is present, the posterior surface of the bodies of the vertebræ, corresponding with the angle, is often bent, rough, and eroded, and the cord over it is apt to become softened, though there may be no compressing growths or thickenings of the membranes.

Thus it happens that the paralysis in these cases may be variously produced. And seeing that it is oftener due to pressure by inflammatory products than to pressure or irritation from the diseased bones themselves, we may the better understand the fact that occasionally a great improvement may set in and become established in regard to the paralysis, although the angular curvature of the spine, and therefore the distortion of the spinal canal, remains as obvious as it ever has been.

In *vertebral caries* with commencing pressure upon the spinal cord, the symptoms will be different, according to the part of the column implicated. The affection is frequently ushered in by an abiding pain in the spine and parts adjacent, often supposed to be 'rheumatic' in nature. Such pains commonly disappear when the patient is in the recumbent position, except during the acts of sneezing or coughing. They are frequently induced by particular kinds of movements, which are more or less difficult on this account. There is also some weakness in the lower part of the body and in the lower extremities. The mere 'weakness' may continue for weeks or even months before there is anything like actual paralysis; though at last this may show itself somewhat abruptly. The patient now becomes unable to stand, though he can still move his legs slightly while lying in bed. At this stage sensation is little, if at all, interfered with; but there may already be some increase in the readiness with which the knee-reflex manifests itself, and ankle-clonus may also be easily obtainable. Next there may be startings of the limbs, and commencing rigidity of the muscles when passive movements are attempted; followed, after a time, by a more marked rigidity (which, when present in the leg muscles, may prevent the manifestation of ankle-clonus and of the knee-jerk).

Later on, if pressure increases, and especially where a complete transverse softening becomes established, sensibility in its various modes becomes implicated. At this period the exaltation of the reflexes often diminishes. For a time the degree of impairment of sensibility and the freedom with which knee-reflex and ankle-clonus may be obtained fluctuates. Meanwhile, painful spasmodic contractions of the legs (with flexion of hip and knee joints) become habitual, persisting through day and night with only rare intermissions.

Although there is some general wasting of the muscles, together with a flabby condition when they are relaxed, they still react almost normally to the faradic current. The skin is often-dry and scurfy. The temperature of the limbs is generally slightly lower than normal.

At the first onset there may be for a few days a difficulty in voiding the urine, but this power soon returns and often continues long after the limbs have become powerless. The bowels are perhaps somewhat constipated, but there is no incontinence of fæces, unless diarrhœa supervenes from any cause, or except when the reflex activity of the bowels is greatly exalted under the influence of aperient medicines.

The above condition of things may last long, without much variation. But, after a time, there will be a gradual mitigation of the symptoms, or the reverse. In the latter case, loss of voluntary control over the bladder and rectum appears; and (especially when sensibility of the body and limbs becomes impaired) the tendency to the formation of sloughs and gangrenous bed-sores becomes increased. With these conditions other complications, such as cystitis, blood-poisoning, etc., may appear and greatly aggravate the condition of the patient, helping to bring about a more speedy termination.

The **diagnosis** of the paralysis associated with *vertebral caries* depends upon the recognition of this causal condition, which, in the early stages, is often a matter of some difficulty. Much will depend upon the existence of pain in particular regions of the spine, or radiating therefrom; of pain which is relieved by the recumbent position, and greatly aggravated by coughing, sneezing, or stooping movements of different kinds (see H. Marsh in 'Brit. Med. Journ.', Vol. i., 1881, p. 913). And yet, in the absence of signs of caries, or of a scrofulous habit of body or history, or of an exciting cause for caries, in cases where there may be little or no prominence of vertebral spines, and even no pain from firm pressure or the application of a hot sponge, we may be helped in our diagnosis of the existence of caries by the distinctive characters of the paralysis itself, namely, its implication of motility principally, the exaggeration of the tendon-reflexes, the more or less marked rigidity of the legs, and the continuance of control over the bladder and rectum.

In cases of the latter type, or where there is only a slight prominence of two to four vertebral spines, it may be difficult, however, to establish a diagnosis between caries and *cancer* of the bodies of the vertebræ. It is true that a rounded prominence of several vertebral spines is met with in cancer more frequently than the angular projection commonly associated with caries, yet this single character will not always aid us; we must look also to the presence or absence of very severe pains, to the clinical grouping of symptoms generally, and to the history of the patient.

The positive diagnosis of the other causes of slow compression of the cord to which reference has been made (exostoses or meningeal tumours), is usually a matter of extreme difficulty. We must be guided by probabilities based upon other associated states or conditions that may be recognisable in our patient, and also by the mode of onset of the affection.

SPINAL PARALYSES OF CHRONIC ONSET.

Under this head we have paralysis resulting from the following causes :—

18. Cancer of Vertebræ.
19. Progressive Muscular Atrophy.
20. Pseudo-hypertrophic Paralysis.
21. Cervical Hypertrophic Pachymeningitis.
22. Tumours and Adventitious Products in the Spinal Meninges.
23. Tumours in the Spinal Cord.

It should be borne in mind that No. 17, *Scrofulous Pachymeningitis, may occasionally have a Chronic Onset, and that Nos.* 19-23 *inclusive may, at times, present a Very Chronic Onset (see p.* 562*).*

18. CANCER OF VERTEBRÆ.

This is an affection distinctly rarer than Pott's disease. Again, it occurs more especially in the latter half of life. According to Charcot it is rarely primary, but is met with principally in the course of scirrhus of the mamma. It may, however, be consecutive to renal or gastric carcinoma; or it may spread directly to the vertebral column from cancer of some of the pre-vertebral lymphatic glands.

Small masses of cancerous growth developed in the bodies of the vertebræ in small number, may remain absolutely latent.

At other times, the bodies of two or three vertebræ become more extensively infiltrated with the new growth ; the softened vertebræ are then apt to become flattened out by the weight of the upper part of the spinal column. In some cases, this flattening of the bodies occurs without producing any appreciable deviation of the vertebral spines ; at other times, it leads to a slight rounded projection in the corresponding region of the spine, which is often quite different from the kind of projection ('angular') commonly met with in Pott's disease.

But the transverse processes and arches of the vertebræ are themselves liable to be infiltrated, and consequently softened, by the cancerous growth. One important consequence of this is, that the nerve roots and trunks become compressed as they pass between the transverse processes, owing to these parts of the softened vertebræ yielding to the superincumbent pressure.

The latter phenomenon leads to the most characteristic features of this disease, in which marked effects are produced by pressure upon the nerve-roots issuing from the vertebral canal, before the spinal cord itself is at all pressed upon. Among these effects, the production of pain is so prominent that Charcot has proposed to designate the disease '*paraplégie douloureuse des cancéreux.*'

The pains due to pressure upon the nerves are commonly very severe, and radiate into the limbs and around the trunk, in accordance with the different distributions of the particular nerves involved.

The pains are more or less continuous, though often exacerbated, and, at times, remitting altogether. They are generally of a burning or otherwise agonising character. Eruptions of herpes may, from time to time, occur in the parts to which the pains are referred. Patches of more or less complete anæsthesia may also occur in different regions, while other portions of the skin become markedly hyperæsthetic. The pains are often greatly aggravated by the least movement.

Again, in consequence of the simultaneous compression of motor fibres we may have spasms or a more or less marked weakness of the muscles innervated by these nerves, and after a time some amount of muscular atrophy with contracture may show itself.

At first there is paresis rather than paralysis; the limbs can be moved pretty freely in bed, and if the patient does not attempt to stand or sit up, it is from dread of the exacerbation of pains which either of these acts may induce.

Symptoms of this type may last for some months before the spinal cord itself begins to be affected, by the cancerous growth pressing upon it after it has involved the dura mater. Thereafter, we may have paralysis with rigidities somewhat similar to what is met with in the paralysis that goes with 'Pott's disease.' There is also the further similarity that pain is commonly felt in the affected vertebræ, especially on percussion or when pressure is made over their spinous processes.

From the point of view of diagnosis it must, however, be borne in mind that on rare occasions, it happens that cancer of the vertebræ does not lead to the softening of the transverse processes, so that there may be neither pressure upon, nor irritation of, the nerve roots, preliminary to the pressure upon or involvement of the spinal cord by the new growth. In these latter cases, the difficulty of diagnosis from meningeal tumour, or even in some cases from Pott's disease, is notably increased, because altogether the most typical and distinctive symptoms of cancer of the vertebræ are those due to pressure upon the spinal nerves and nerve roots.*

It is always of great importance in any case in which the possible existence of cancer of the vertebræ suggests itself, to examine the body generally for evidence of cancer in other parts, and to bear in mind that scirrhus of the mamma seems particularly apt to be followed by cancer of the vertebræ.

An aortic aneurism or an hydatid cyst may at times give rise to pains very similar in character to those associated with cancer of the vertebræ, nor must 'osteomalacia' be altogether lost sight of.

Again, 'hypertrophic cervical pachymeningitis' may cause pains very similar to those that would be caused by cancer of the vertebræ in the cervical region—a situation, however, in which cancer is decidedly less frequent than it is in the lumbar or dorsal regions.

Finally, it must not be lost sight of that cancer of the vertebræ, at any level, may occasionally give rise to unilateral rather than to bilateral pains, owing to pressure occurring only upon the nerves of one side.

19. PROGRESSIVE MUSCULAR ATROPHY.

This is a comparatively common disease, in one or other of its forms. It is also known as 'Cruveilhier's Palsy,' or 'Wasting Palsy.' It is essentially a disease of middle life; it occurs most commonly between the ages of thirty and fifty years. It is also much more common in males than in females, the proportion being about five or six to one. In childhood we have to do with what is by many believed to be an allied affection, viz., pseudo-hypertrophic paralysis.

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* It should be said that in these cases of cancer of the vertebræ in which the nerve roots and their ganglia are pressed upon, Charcot has found the related nerve trunks swollen and redder than natural—the irritation has, in fact, induced in them a distinct inflammatory condition. The pains, again, associated as they are with trophic lesions, are commonly spoken of as 'pseudo-neuralgic' in character, though this term seems to suggest a distinction of doubtful value.

Concerning both of these diseases an immense amount of discussion has taken place as to whether they are to be regarded as intrinsic diseases of muscle, or whether they are primarily diseases of the spinal cord causing atrophy of muscles as sequential phenomena. Although there are many reasons for believing in the essential similarity of the two affections (a view which I have myself been always inclined to hold), it must be confessed that the development of our knowledge concerning them has, of late, by no means tended to strengthen this notion. Thus, the general consensus of opinion has recently been strongly setting in the direction that 'pseudo-hypertrophic paralysis' is not associated with any constant changes in the spinal cord, and that it is, therefore, due rather to primary degenerative changes in the muscles While, on the contrary, the general agreement is now even stronger to the effect that in the case of 'progressive muscular atrophy,' we have to do with definite primary changes in the spinal cord, and with secondary atrophies in muscles.

In progressive muscular atrophy, in fact, we have atrophies of the great ganglion cells in different parts of the anterior cornua, varying in situation according to the different forms in which the disease presents itself, though always most common in the cervical region. The disease of the nerve cells seems to be due to some chronic degenerative process, causing ganglion cells here and there to atrophy, while other contiguous cells may show no change whatsoever. The fact that individual cells here and there undergo this atrophic process, and not a whole group of such cells simultaneously, goes far to account for the clinical differences met with between this disease and 'chronic spinal paralysis.' The result of such different distributions of neural atrophy being that in 'chronic spinal paralysis' (as also in 'acute spinal paralysis') the whole of the cells in connection with certain muscles become diseased simultaneously, and, as a consequence, the whole of the related muscle fibres become atrophied at the same time; while in 'progressive muscular atrophy' only certain of the cells in relation with a particular group of muscles undergo an atrophic process, to be followed gradually by other sets. Consequently a progressive atrophy occurs also in the muscular fibres in relation with these nerve cells, while the remaining muscular fibres of the same muscle retain their healthy condition and, as a consequence, present normal electrical reactions.

Of course, in all these cases the motor nerves intervening between the atrophied nerve cells and the atrophied muscle fibres likewise undergo an atrophic process. This atrophy of the motor roots was clearly recognised by Cruveilhier, as the most obvious macroscopical change on the side of the nervous system.

Another peculiarity of this disease is also perfectly explained by the progressive mode in which the disease affects particular nerve cells and particular muscle fibres in relation therewith, leaving the remaining contiguous nerve cells and fibres for a time in a healthy condition. The peculiarity to which I allude is the fact that in 'progressive muscular atrophy,' we have not to do (as in the other two diseases above named) with paralysis of certain muscles as a first event and then an atrophic process in such muscles, but rather with atrophy or wasting as the first event associated with a merely proportional paresis as the disease progresses—actual paralysis manifesting itself only as the final event on the completion of the atrophic process. The weakening of the parts affected is what first attracts the notice of the patient, and then he, or the medical attendant to whom he applies, recognises that the parts which are weak are also more or less obviously wasted.

It often happens that the muscles in which this progressive muscular atrophy is advancing, show spontaneous flickerings or 'fibrillations.' Or, if not spontaneous, such fibrillations may often be easily evoked by the application of some cold substance or by gentle mechanical irritation. At times, however, this sign is altogether absent.

The phenomena by which this disease is characterised are, therefore, extremely simple. Increasing paresis together with increasing atrophy of muscles, which still respond freely to faradisation, beginning in certain definite groups and slowly spreading to others (in ways to be subsequently referred to) constitute its distinguishing features. There is an absence of sensory defects, or other collateral phenomena—with the exception that, in a certain class of these cases, in which the onset and progress is rather subacute than chronic, there may be pains in the muscles that are undergoing atrophy, as well as in neighbouring joints.

In regard to the causation of progressive muscular atrophy our knowledge is still very defective. A tendency to the disease is certainly inherited in not a few of the cases, and sometimes this may be traced through several generations. Again, over use of certain groups of muscles seems to favour the occurrence of atrophy in some cases. It seems occasionally to follow falls or concussions of various kinds; or it may succeed some definite exposure to cold and wet; whilst at other times it may follow in the wake of syphilis or some one or other of the zymotic diseases. In another class of cases, however, it sets in without apparent relation to any such or other definite assignable causes, and in these cases especially, enquiry may reveal the existence of some hereditary tendency to the disease.

The following different types of progressive muscular atrophy may be met with :—

(1).—A lower arm type, in which the disease begins in the small muscles of the hand—those of the thenar and hypothenar eminence, together with the interossei—and, in some cases, may limit itself to this region.

The disease in the cord in this case is found in the grey matter of the anterior cornu corresponding with the second dorsal nerve. There is an interesting point concerning the pupil in cases of this type. Ferrier and Yeo have shown that it is only the second dorsal motor root in the monkey which contains the dilating fibres of the iris. Stimulation of this root, as they found, caused dilatation of the pupil and contraction of the intrinsic muscles of the hand (p. 535). Hughlings Jackson refers to two cases of progressive muscular atrophy, where the intrinsic muscles of the hand were affected, and in which there was also smallness of the pupil on the same side and no dilatation when the eye was shaded.

(2).—An upper arm type, in which the deltoid, the supra and infra-spinatus, and other muscles about the shoulder joint are first or principally affected, on one or both sides.

The disease in the cord in this case is found in the grey matter corresponding with the roots of the fifth, sixth, and seventh cervical nerves.

The disease may remain limited to these regions, if not altogether, certainly for very long periods.

(3).—Cases in which, whether the disease begins in the lower or in the upper part of the arm, it soon spreads so as to involve the whole of the muscles of the limb, as well as those connecting it with the scapula and trunk. After a shorter or longer interval the disease in these cases is apt to spread to the trunk muscles generally, as well as to those of the lower extremities, death being ultimately brought about, in the main, through paralysis of the respiratory muscles.

These constitute the most typically progressive forms of the disease. When the atrophy is extreme in the interossei and muscles of the wrist the fingers become partially flexed and contracted in a peculiar manner, so as to produce a fancied resemblance to a bird's claw. This is the *main en griffe* of Duchenne (see Fig. 131, c).

4.—Altogether rarer cases occur in which the atrophy shows itself first in the muscles of the trunk and lower extremities,

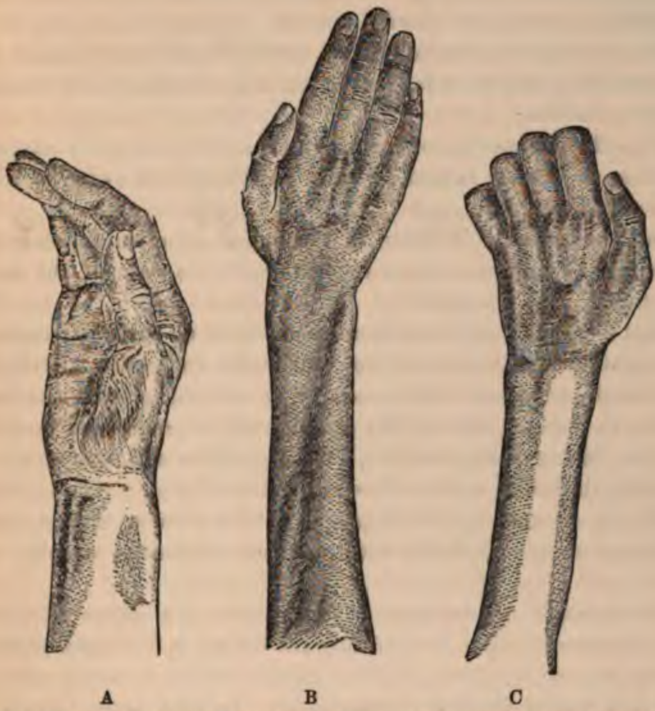

Fig. 131. Hand in a case of Progressive Muscular Atrophy which commenced in the muscles of the thumb [after Duchenne].

B, and C.—Hands, the muscles of which are almost completely destroyed, from a case of Progressive Muscular Atrophy, which had become general at the end of two years [after Duchenne].

or in one or other of these sites. How exceptional these forms of the disease are, however, may be judged from the fact that out of 146 cases collected by Friedreich, 111 commenced in the upper extremity, 27 in the lower extremity, and 8 in the lumbar muscles. Duchenne only saw the disease commence in the lower extremities in 2 out of 159 cases; and in my own experience this mode of commencement has also been extremely rare.

Where the disease begins in the trunk muscles, it sometimes involves the lumbar muscles first and sometimes the abdominal muscles—and each of these forms is marked by a characteristic attitude. The back is much curved forwards in each case, the curvature being most marked, and the lower part of the abdomen the most prominent part of the convexity, in the cases in which the abdominal muscles are first atrophied; whereas, in cases of atrophy of the lumbar muscles, the concavity of the back is less marked, and the maximum projection in front is rather above than below the level of the umbilicus.

(5).—In another class of cases progressive muscular atrophy becomes associated with labio-glosso-laryngeal paralysis (p. 361).

In these cases the affection of the bulbar ganglion cells may precede, occur simultaneously, or follow towards the close of the spinal affection.

It is a peculiar fact that there seems to be no particular tendency for 'ophthalmoplegia externa' to occur in this connection, although it is generally supposed to be caused by an atrophic process essentially similar to that met with in the spinal cord in progressive muscular atrophy, taking place, however, in the ganglion cells composing the nuclei of the ocular motor nerves in the floor of the Sylvian aqueduct. Curiously enough, ophthalmoplegia externa occurs, at times, with locomotor ataxy rather than with progressive muscular atrophy.

Notwithstanding the name progressive muscular atrophy, it is important to bear in mind that the disease is not necessarily and fatally progressive. It may limit its manifestations to certain muscles, and then become arrested spontaneously. In other cases, though the disease is distinctly progressive for a time, its advance may, after a while, perhaps under the influence of treatment or improved conditions of life, come to a standstill.

The atrophy which has been caused by over-use of the affected group of muscles is very likely to limit itself; while, on the other hand, where we have to do with an hereditary tendency to the disease, the disease itself is especially prone to become generalized.

Again, it is important to remember that in certain exceptional cases the disease begins in a sub-acute manner, and may be associated with so much pain and tenderness in joints as to be mistaken for sub-acute rheumatic fever. I have seen one remarkable case of this type in which, with an antecedent history of syphilis, there was much exposure to wet and cold, speedily followed by marked joint pains and tenderness and, in the course of two or three weeks, the most notable muscular atrophy of nearly all the muscles of the upper extremities and of those connecting it with the scapula and trunk. Under active treatment, this man, in the course of a few weeks, greatly improved. The morbid process ceased to spread and some power was regained over the greatly weakened limbs. This same patient was seen, about two years afterwards, in substantially the same condition as when he left the hospital, no other muscles had become involved and those which were atrophied still freely responded to faradisation, as they had done all along. Pains in the muscles and joints are most apt to occur in cases in which the disease has been initiated by exposure to wet and cold.

Progressive muscular atrophy can be **diagnosed** usually with the greatest ease, if we bear in mind its slow and progressive character, the fact that atrophy precedes paralysis, and that the atrophied muscles (so long as the wasting is not in its final stages) still respond freely to the faradic current. These characters would suffice to prevent our confounding most other forms of disease with progressive muscular atrophy, and amongst them, that rare form which is caused by complete paralysis of the ulnar nerve, where also there is the production of the 'clawed hand' or 'main en griffe,' though with the peculiarity that the deformity is most marked in the ring and little fingers, in which also there is more or less impairment of sensibility.

20. PSEUDO-HYPERTROPHIC PARALYSIS.

It is very doubtful whether this disease ought to be regarded as an affection of the spinal cord at all. Certainly the spinal cord has been found to be free from appreciable morbid changes in several typical cases in which it has been carefully examined by highly competent observers. The evidence has, of late, been increasing, which tends to show that it is rather a malady dependent upon intrinsic disease of the muscles. The interstitial connective tissue of the muscles undergoes a more or less extensive process of hyperplasia, many of the new formed connective tissue cells becoming converted into fat cells, while the new formed tissue strangles, and leads to the gradual atrophy of, the muscular fibres themselves.

Even though the malady may have no strict title to a place among diseases of the spinal cord, it will be convenient briefly to refer to it here, since its diagnostic relations are certainly with these affections.

As in progressive muscular atrophy, so in pseudo-hypertrophic paralysis, we have to do not with a real paralysis, but rather with a progressive paresis that increases *pari passu* with the atrophy occurring in the muscular fibres themselves.

The disease is hereditary and is much more frequently transmitted through the female than through the male side of the family, though it is actually much more common in boys than it is in girls. Nothing else concerning the etiology of the disease can be said to be known. It is only very rarely that cases are met with in adults.

According to Gowers, out of 139 recorded cases, 123 were males and only 16 females. He found that one-half of the male cases had occurred before the sixth year, and about three-fourths of them before the tenth year. In the females, however, one-half of the cases commenced after the tenth year.

FIG. 132. FIG. 133.

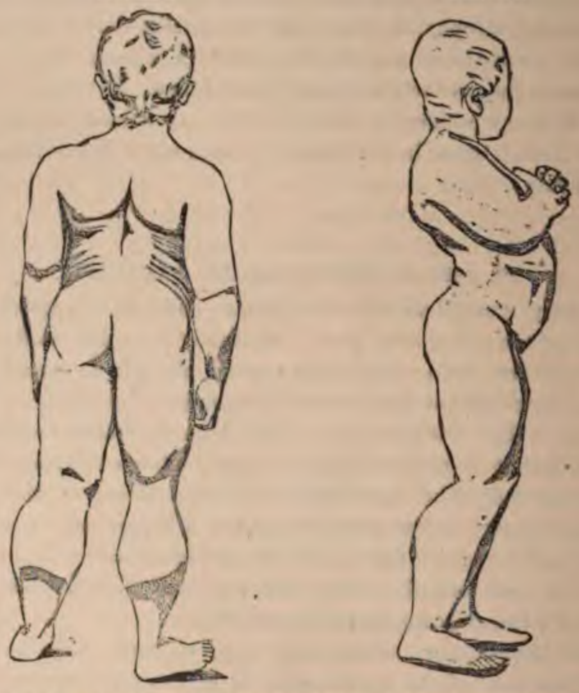

FIG. 132. BACK VIEW, AND FIG. 133 SIDE VIEW OF A CASE OF PSEUDO-HYPERTROPHIC PARALYSIS, IN WHICH ALMOST ALL THE MUSCLES OF THE BODY WERE HYPERTROPHIED [after Duchenne].

The first symptoms of the disease consist in weakness of the lower extremities, together with an unsteady, stumbling gait.

After such symptoms have increased for a time the child adopts a characteristic attitude and gait. He mostly stands with his feet wide apart and his heels slightly raised; the belly is very prominent and the back is much curved in the lower dorsal and lumbar region, while the shoulders project backwards. The condition is one of very unstable equilibrium. The gait is waddling, awkward, and very unsteady.

He raises himself from the floor with considerable difficulty and by several stages, in the last of which the patient gradually straightens his body by the process described as 'climbing up his thighs' (Fig. 134).

When the patient is stripped at this stage of the disease, the calves and buttocks are often found to be distinctly enlarged (hence the name of the disease) and notably harder than natural. Muscles in the upper part of the body are much more rarely hypertrophied. It is distinctly more common to find them atrophied. This atrophy, according to Gowers, is especially noteworthy and constant in the latissimus dorsi and in the sterno-costal portion of the pectoralis major.

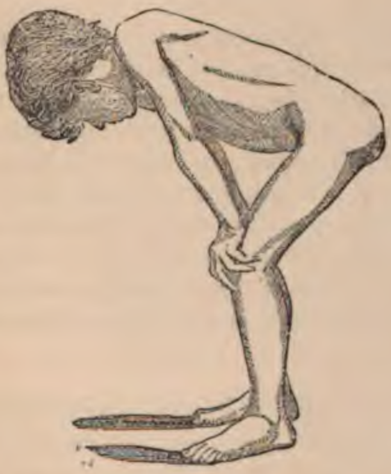

FIG. 134. CLIMBING UP THE THIGHS IN PSEUDO-HYPERTROPHIC PARALYSIS. The figure represents a boy in the late or atrophic stage of the disease [after Gowers].

The tendency is for all the muscles of the body ultimately to undergo a degenerative and atrophic process, so that a general powerless condition is ultimately induced. At this late stage of the disease, even the muscles of the lower extremities, which during earlier stages were hypertrophied, become diminished in bulk, so that we have everywhere evidences of atrophy of muscle.

But for the fact that occasionally the children in whom these changes develop show more or less marked signs of mental deficiency, this disease has no other independent characters. The irritability of the muscles to faradisation is usually distinctly diminished, and the knee jerks are abolished; these characters are, however, dependent upon the changes in the muscles themselves.

The sensibility of the limbs and body remain intact. The functions of the bladder and rectum are not interfered with, except possibly in the final stages, or in those cases in which from the beginning the disease has been associated with distinct mental defects.

The **diagnosis** of the affection, in the majority of cases, is not attended with any difficulty, provided the child be stripped and carefully examined, and the medical attendant is aware of the existence of an affection in which progressive weakness and difficulty in walking is associated with an apparent hypertrophy of some of the muscles of the lower extremities.

The hypertrophic condition of the calves and buttocks is, however, by no means equally well marked in all cases; in some instances, indeed, no appreciable enlargement exists, but even then the muscles are generally harder or firmer to the touch than natural. In such cases Gowers calls special attention to the diagnostic importance of the distinct atrophy which is so frequently recognizable after a time in the latissimus dorsi and the lower part of the pectoralis major. The existence of atrophy of other muscles, also, together with the slow onset and progress of the case, and possibly the family history, may likewise aid to confirm the diagnosis of this peculiar disease.

21. CERVICAL HYPERTROPHIC PACHYMENINGITIS.

This disease has been most fully recognized and described by Charcot and Joufroy, though a similar condition seems to have attracted some attention from earlier observers, who, under a mistaken impression, spoke of it as 'hypertrophy of the spinal cord.'

In this disease the dura mater becomes extremely thickened and hypertrophied (sometimes to the depth of half an inch) by a chronic inflammatory process resulting in a fibrous hyperplasia, which affects the whole of its circumference, and also involves the arachnoid and pia mater for a varying longitudinal extent, in some particular region of the cord.

Although it may occur in other regions such a process is by far the most common in the membranes surrounding the cervical enlargement of the cord. The greatly thickened membranes not only irritate and compress the nerve roots as they pass through it, but after a time they lead to marked compression (followed mostly by induration) of the included portion of the spinal cord, changes which are commonly spoken of as a 'chronic transverse myelitis.'

Sometimes the disease progresses in a rather irregular manner, though more frequently it advances by definite stages. Charcot divides it into two periods—(1) a stage of irritation, associated with much pain, of comparatively brief duration (two to four months); and (2) a stage of paralysis with muscular atrophy.

(1).—During the first stage of the disease symptoms due to irritation of the sensory nerves predominate. The patient complains of severe neuralgic pains in the back of the neck, extending upwards, and also spreading into the shoulders and upper extremities.

The pains are accompanied by a kind of rigidity, especially in the muscles of the neck, which causes the head and neck to assume a fixed and constrained aspect very similar to that met with in those who suffer from cervical caries.

The pains are more or less continuous, but subject to exacerbations from time to time. Though felt in the joints as well as in other parts, these structures do not commonly enlarge. Besides the severe pains, formications and numbness are also experienced in the upper extremities. Trophic disturbances occasionally make their appearance in the form of herpetic or other eruptions.

During this period there is usually some amount of paresis of the upper extremities.

(2).—In the second stage of this malady we have to do with a more profound implication of the peripheral nerves, as well as with increasing pressure upon, and changes in, the affected region of the spinal cord.

A great change takes place in the symptoms; the pains gradually cease, while the paresis deepens into actual paralysis, the muscles at the same time undergoing an atrophic process pretty equally throughout the whole extremity—after the fashion that prevails in amyotrophic lateral sclerosis.

Still, it is most common for the paralysis to predominate in the muscles supplied by the ulnar and median nerves, and for the atrophy also to be most marked therein. The muscles supplied by the musculo-spiral nerve are decidedly less affected. This, at least, is what happens in the majority of cases, that is, when the lower half of the cervical enlargement is the region most distinctly involved in the morbid process. The result of the above-mentioned predominance of paralysis in certain groups of muscles is the production of a peculiar position of the hand (Fig. 135), in which the hand and the thumb are strongly extended while the fingers are flexed in claw-like fashion.

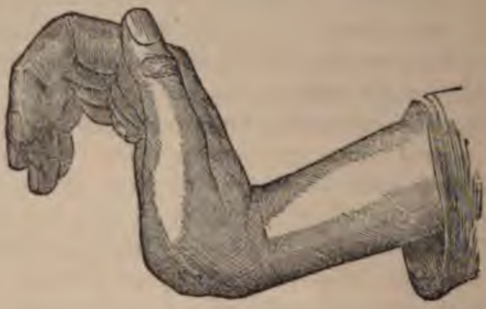

FIG. 135. ATTITUDE OF THE HAND IN CERVICAL HYPERTROPHIC PACHYMENINGITIS WHEN THE DISEASE IS ON A LEVEL WITH THE LOWER HALF OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD [after Charcot].

When the stress of the disease occurs, however, at a higher level, that is, opposite the upper half of the cervical enlargement, then it is that the roots of the musculo-spiral nerve, or the corresponding areas of grey matter, become more involved than those of the ulnar and median nerves, and the maximum amount of paralysis also occurs in muscles supplied by the former nerve. The result, as was seen in a remarkable case under the care of Dr. Leech, was to produce an altogether different attitude of the hand and arm. Speaking of this case, Ross says:—"The arm is held close to the side, the forearm is extended on the arm and strongly pronated, the hand is flexed on the forearm, the fingers are on a line with or only slightly extended on the metacarpal bones, and the phalanges are extended upon one another, while the thumb is flexed into the palm." (Fig. 136.)

The paralysed and atrophied muscles either wholly, or almost

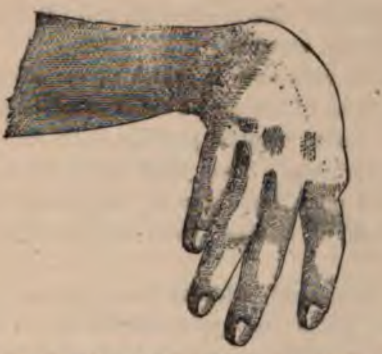

FIG. 136. ATTITUDE OF THE HAND IN CERVICAL HYPERTROPHIC MENINGITIS WHEN THE LESION IS SITUATED ON A LEVEL WITH THE UPPER HALF OF THE CERVICAL ENLARGEMENT OF THE SPINAL CORD [after Ross].

wholly, fail to respond to the faradic current, and, after a time, contractures begin to appear in the upper extremities. The skin, also, may become anæsthetic here and there in patches, and some of these patches may extend so as to involve parts of the trunk.

Later on, the lower extremities also become paralysed; the deep reflexes are exaggerated, and, after a time, contractures become developed. The muscles of the lower extremities do not become atrophied to any notable extent.

This paralysis of the lower extremities is doubtless due to the changes in the spinal cord set up by the constricting pressure of the hypertrophied dura mater, as well as to the fact of the pressure itself. Charcot explains the rigidity of the muscles in accordance with his well known views on this subject—that is to say he supposes the 'transverse myelitis' to give rise to descending degenerations of a secondary nature in the lateral columns, and that these degenerations cause the rigidity in a manner to which reference has elsewhere been made (p. 218). I think, however, that the existence of this rigidity is an indication that the conducting functions of the cord are not wholly annulled by the constricting lesion above, and that an excess of cerebellar influence is reaching the lumbar grey matter in consequence of the degeneration of the pyramidal tracts in the lateral columns (p. 222).

In regard to **diagnosis,** there are only three diseases which are at all likely to be confounded with hypertrophic cervical pachymeningitis. These are (1) cancer of the dura mater opposite the cervical enlargement, (2) amyotrophic lateral sclerosis, and (3) Pott's disease affecting the lower cervical vertebræ.

In the extremely rare cases in which cancer affects the dura mater in the cervical region of the cord a set of symptoms may be developed agreeing in the closest manner possible with those of the affection now under consideration. Some years ago I saw a case of this kind; it ran, however, a more rapid course, and death was brought about by the production of a total transverse softening in the cervical region.

In amyotrophic lateral sclerosis we have no preliminary period marked by severe pains and hyperæsthesia in the upper extremities; while, on the other hand, the paralysed and rigid muscles of the lower extremities speedily undergo atrophy in this affection. Again, the course of this latter disease is more rapid, and the medulla oblongata is prone to become affected after the lumbar region of the cord; death following at no distant date. The difference in the course of events is, therefore, great in the case of cervical hypertrophic pachymeningitis, in which we have a period of severe pains in the upper extremities, no atrophy in the paralysed lower extremities, a slow course, no implication of the medulla, and at times an incomplete recovery.

Pott's disease affecting the lower cervical vertebræ may give rise to pains, less severe it is true, but in the same situations as those met with in hypertrophic pachymeningitis, and also to the same kind of fixed attitude of the head and neck. But, in Pott's disease, movements, pressure upon the vertebræ in the affected region, or any percussion of the spinal column, is apt to give rise to much more pain than could be similarly produced in pachymeningitis. Again, in Pott's disease, the patient is more likely to be of scrofulous type and young, than of middle age. Of course, if there should be anything like a definite angular curvature distinguishable, no such difficulty of diagnosis as that to which we are now referring would ever arise.

22. TUMOURS AND ADVENTITIOUS PRODUCTS IN THE SPINAL MENINGES.

Under this head are included various different kinds of new growths or adventitious products. The nature of the growths to be met with, constituting the pathological side to the question, has already been referred to elsewhere (p. 546), so that not much remains to be added here.

In diagnosing the presence, as well as the nature, of certain tumours in the spinal meninges, we may, in suitable cases, be influenced by the existence of a *syphilitic history*, by evidence of *cancer* in other parts of the body, or by the existence of a markedly *scrofulous habit of body*. In either of these cases, what determines the appearance of a growth of this or that type in the spinal meninges, rather than in any other site, generally remains in as much obscurity as when the growths are solitary or of non-diathetic origin. Among such determining or exciting causes, only one of those usually cited appears to be of real potency, namely, the occurrence of blows upon the spine, or injuries of various kinds. These seem, at times, to be the immediately exciting cause of the development of a new growth in the spinal meninges.

The occurrence of *sarcomatous tumours* in other parts of the body, or the evidence of the existence of *hydatids* in the liver or elsewhere, are facts that may at other times guide our diagnosis as to the nature of a supposed extra-medullary tumour. That is the kind of evidence upon which we are forced to rely generally; still, in the case of a hydatid cyst developing in the tissues outside the spinal meninges, should any portion of the sac, after a time, project outside the spinal canal, important information may be derived from an examination of the back, and from the presence at the side of the spine of an elastic fluctuating swelling. The puncture of such a swelling with a small syringe, and the drawing away of mere hydatid fluid, even without the discovery of echinococci or their hooklets, ought to suffice to convert our surmises into certainty. The hydatid fluid has characters of its own, which are generally quite distinctive. Such a diagnosis is only likely to be made with the greatest rarity in this country; but the occasion for making it might recur with decidedly greater frequency, in countries such as South Australia, where hydatid disease is very common.

There is one other pathological condition that may occasionally give rise to pressure upon the spinal cord from without, and that is *aneurism of the aorta* after it has eroded parts of the bodies of one or two vertebræ. Such a cause as this, however, is likely to be diagnosed long before it comes to the stage when it may produce symptoms simulating those of a tumour of the spinal meninges.

In all the cases to which reference has been made there would be a certain generic similarity in the symptoms produced. These symptoms comprise two more or less distinctly defined stages.

The first stage is represented by symptoms referable principally to irritation of the roots of spinal nerves and of the meninges; while, in the second stage, we have to do with symptoms produced by pressure upon the spinal cord itself.

Stage of Irritation.—This stage is, in different cases, very variable in its duration, in accordance with differences in the size and rate of growth of the tumours or adventitious products developing in the spinal meninges. It may last only for several months, or, in other cases, even for two or three years.

Its most typical symptoms are pains in the back at the site of the new growth, together with pains in the areas of distribution of the sensory nerve roots that happen to be pressed upon or irritated. In these same areas there may be patches of hyperæsthesia or of anæsthesia; or the two conditions may coexist in different parts. These areas of sensory disturbance will be situated on the trunk, or on the limbs, or on both, according to the varying longitudinal situation of the growth. Evidences of irritation of motor roots may likewise exist, subject to similar variations in site; and in both cases the morbid symptoms may be more marked on one side of the body than the other, or may be wholly confined to one side. The signs of irritation of the motor roots occur in the form of twitchings or rigidity of some of the muscles in connection with such roots. In rare cases it may happen, where the nerve root becomes more severely pressed upon, that a localised paralysis (of a partial type) may occur in particular groups of muscles (see p. 532) associated with partial muscular atrophies.

In some cases, symptoms of this type are well marked, in others they are apt to be obscure and ill-defined.

Stage of compression of the Spinal Cord.—At this stage, also, great variations in the symptomatology occur, especially near its commencement, in accordance with the direction from which the pressure first comes—that is, whether the posterior columns, or the anterior, or the lateral columns of one side are the parts which are first pressed upon. Other differences will be found to be dependent upon the longitudinal situation of the compressing body—roughly speaking, whether it has a cervical, a dorsal, or a lumbar site.

Later on, the early and somewhat indefinite symptoms produced by the tumour as a compressor of the spinal cord may become more and more defined, so as, at last, to produce a paraplegic condition of a pretty complete type. Before it becomes complete, such a paraplegia is often associated with distinct rigidities, together with exaltation of the deep reflexes; sensibility also may be much impaired, or there may be hyperæsthesia in certain regions. An incomplete paraplegia of this type may be caused, in the main, by the actual compression of the spinal cord; though, where such pressure is well marked, the symptoms are apt to be soon aggravated by secondary pathological changes set up in the spinal cord itself. These may be of an atrophic indurating nature—changes principally of a degenerative type—such as are most prone to occur when the compressing cause operates very slowly.

At any time, however, in the history of such a case, the symptoms are apt to assume a more severe type owing to the establishment of a complete paraplegic condition consequent upon the setting up of an acute inflammatory, or a non-inflammatory, softening at the seat of compression involving the whole thickness of the spinal cord. We have then produced all the symptoms pertaining to a 'total transverse softening' (see p. 580), including before long the occurrence of bedsores, with inflammation of the bladder and other complications—through one or other of which a fatal termination may at last be brought about.

The **diagnosis** can only be made after the fullest consideration of the patient's condition, both general and local. The principal diseases with which tumour of the spinal meninges is most apt to be confounded are cervical hypertrophic pachymeningitis, cancer of the vertebræ, and the pachymeningitis that goes with Pott's disease. For aid in establishing such a diagnosis see Table IV., p. 564. The diagnosis may generally be arrived at when the probability of the existence of either of the other diseases belonging to Group C can be excluded.

23. TUMOURS OF THE SPINAL CORD.

Tumours originating in the substance of the spinal cord may be regarded as belonging to two classes, according as they represent (*a*) mere local accidents in the form of perverted tissue changes; or (*b*) such local accidents developing under the influence of a distinct general diathetic state, such as syphilis or scrofulosis.

(*a*). Of the purely local overgrowths, the most typical, and perhaps also the most frequently occurring, are *gliomata*. In such a tumour we have an exuberant overgrowth, as Virchow and most other pathologists suppose, starting from the neuroglia of a certain portion of the cord. At first the growth infiltrates and substitutes itself in the place of a certain amount of nerve-tissue; but it soon becomes excessive in quantity (spreading in area perhaps at the same time), and thus comes to exercise a more and more marked compression upon the remaining tracts of nerve-tissue composing the cord enclosed, as it is, within the narrow and unyielding boundaries of the spinal canal. These gliomata are oftentimes extremely vascular. They are liable to undergo a certain amount of central softening; and into their substance, especially in the softened foci, hæmorrhages are very apt to occur. Softening of nerve-tissue may also, at a certain stage, take place around the growth, and possibly extend for a variable distance above and below.

Other tumours of an allied nature, such as *sarcomata* and *myxomata*, also at times develop, either in their pure types or with blended characters, within the spinal cord. They present few intrinsic peculiarities in their manner of affecting the cord. They rarely attain any large size; indeed the limitations in the diameter of the spinal canal only permit of much increase in one direction. Elongated tumours of the kind indicated are occasionally met with. To a considerable extent, such tumours have an infiltrating mode of growth, though their boundaries are apt to be rather more defined than those of gliomata.

In regard to the causes of these tumours, almost nothing more definite can be said than that they seem, at times, to find occasion and conditions suitable for their initiation after some blow upon the spine, or concussion of the spinal cord. In other cases, no exciting causes of any kind can be defined.

(*b*). Of the growths which tend to occur in the spinal cord (as occasionally in other parts of the body) under the influence of some general state or diathetic condition, two are especially to be named. These are *scrofulous growths* ('tubercular'), and *syphilitic gummata*. The former are generally small, varying in size from a mustard seed to a pea, and only very rarely attaining the dimensions of a hazel-nut. Next to gliomata they are the new growths most frequently met with in the substance of the spinal cord. When small, they may occur in association with a cerebro-spinal tubercular meningitis; but at other times they are found, and especially the larger growths, existing independently of any acute inflammation of the meninges. In this latter case, the tumours may be combined with a certain amount of adjacent and secondary softening of the substance of the cord.

Syphilitic gummata, originating in the cord itself, occur only with the greatest rarity. They are more frequently found starting from the meninges, and then they may press upon, or actually grow into, the nerve-substance.

Cancer is believed not to occur primarily within the spinal cord, though it may grow into its substance (or seriously press upon it) when it originates either in the meninges or in one of the vertebræ.

The difficulties of diagnosis are almost always very great in the case of tumours of the spinal cord, because, in their early stages and occasionally for prolonged periods, they are associated with slight and somewhat vague symptoms.

Independently of the variations in different cases consequent upon the longitudinal situation or level of the tumour in the spinal cord, the symptoms to which they give rise in various parts of the body may be more or less vague anomalies of sensibility in different regions, associated with a certain amount of paresis, rarely amounting to actual paralysis.

Growths from the meninges, or from the vertebræ, pressing upon the spinal cord, are not quite so apt to run a latent course for any length of time, since they are rather more prone to involve the anterior or the posterior roots on one or on both sides—at first irritating them, and subsequently causing paralysis from pressure. Thus localised numbness, pains, or anæsthesia (either alone or associated with twitchings, cramps, or paralysis) confined to certain parts of the body, are rather more common incidents during the growth of extra than of intra-medullary tumours. Still the diagnosis between these two classes of tumours may in some cases be impossible.

Sclerosis in its 'insular' form, especially when the patches are few or close together, may also present symptoms almost inseparable from the first stage of some intra-medullary tumour. The important fact is, however, that insular sclerosis in the cord tends to become more and more generalised, and thus gives rise to a proportionately widening range of symptoms; otherwise it limits itself to special columns, and thus becomes associated with more definite sets of symptoms.

With any of these tumours of the spinal cord, the symptoms are, after a time, liable to undergo a sudden and grave increase, owing to the occurrence of a hæmorrhage into its substance (and perhaps into adjacent regions of the spinal cord), or else owing to the commencement of a process of secondary transverse softening. Beyond these possibilities of sudden grave augmentation of symptoms, the course of intra-medullary tumours is also apt to be marked by peculiar exacerbations and remissions from time to time, in association with periods of altered growth or vascularity of the tumour itself.

The very gradual onset of the symptoms in cases of tumour of the spinal cord, is a point of great importance in the **diagnosis** of these conditions. Thus, for instance, we eliminate arachnoid or intra-medullary hæmorrhages, and also the numerous class of cases of softening of the spinal cord, with other affections having a more or less abrupt origin. The diagnosis of tumour of the cord as distinct from its compression by disease of vertebræ (where there is also generally a slow evolution of paralytic symptoms), must be based in part upon the absence of any evidence of vertebral disease. The diagnosis from meningeal tumours has already been referred to; and so also has the diagnosis from mere sclerosis of the spinal cord, in which the connective-tissue overgrowth is not sufficiently bulky to amount to an actual tumour.

If the arrival at a diagnosis as to the existence of a tumour of the spinal cord is a process beset with difficulties, these by no means cease when, passing from the primary, we have to approach the secondary question as to the *nature* of the growth presumed to exist. But little is possible in this direction.

It is true that, with a history of pre-existing syphilis, even without the evidence of other simultaneous manifestations, we should be warranted in assuming it to be quite possible that an existing growth in the spinal cord is syphilitic in nature, and in treating the patient accordingly. The presumptions in favour of the tubercular or scrofulous nature of a supposed new-growth in the spinal cord would rarely carry with them more than a moderate amount of cogency. Still, occasionally, the general habit of the patient, together with the fact of the existence of scrofulous enlargement of glands, or of some form of phthisis, might give more or less probability to such a conclusion. Beyond this, not much can be done in the way of diagnosing special kinds of tumours. We may be guided in our opinion as to the possible existence of a sarcoma by the presence of one or more of such growths in other parts of the body; or, failing this, we may recollect that primary cancer affecting the spinal cord is almost unknown, and that gliomatous tumours are, next to the tubercular or scrofulous, those which are most frequently met with in the cord itself.

SPINAL PARALYSES OF VERY CHRONIC ONSET.

Under this head we have paralyses resulting from the following causes :—

24. Primary Lateral Sclerosis.
25. Amyotrophic Lateral Sclerosis.
26. Locomotor Ataxy.
27. Insular or Disseminated Sclerosis.
28. Friedreich's Disease (Diffuse Sclerosis).

It should be borne in mind that Nos. 19-23 *inclusive may occasionally cause paralyses of Very Chronic Onset (see p.* 562*).*

24. PRIMARY LATERAL SCLEROSIS.

This disease is also known by the name of 'Spasmodic Spinal Paralysis.'

It is a malady which is distinctly more common in males than in females, and it occurs in the majority of cases in adults from twenty to fifty years of age. Erb and others have also described spasmodic forms of paralysis occurring in children. Some of these are possibly instances of the disease now under consideration, though others of them are rather due to some primary destructive disease in the Rolandic areas of the cerebral cortex, or to some developmental defect in the same situation as Ross ('Brain,' Vol. i., p. 447) has shown. Some of the cases included under the term 'spastic paraplegia of infancy,' seem undoubtedly to be caused in this way (see also Hadden, in 'Brain,' Vol. vi.).

On the other hand I once met with what, at first, appeared to be a case of lateral sclerosis in a child about ten years of age, though a more thorough examination subsequently showed that lateral sclerosis was only a prominent part of what was really a 'multiple sclerosis' of cerebro-spinal type.

In some cases, the disease appears independently of any appreciable predisposing or exciting causes ; but, in other instances, falls or other traumatic influences seem to be distinctly connected with its origin. On rare occasions, exposure to wet and cold has seemed to have some influence over the genesis of this, as well as over so many other forms of, spinal disease.

This disease often sets in almost imperceptibly, and the symptoms continue to develop in a very slow and gradual manner.

Patients begin to complain first of mere weakness of the lower extremities, and this continues to increase till a well-marked condition of paresis exists. There is great difficulty in getting upstairs, and the feet begin to drag even when the patient walks on level ground. This paresis may soon be associated with more or less of muscular twitchings, either painless or painful in character—often more marked in the morning, but sometimes worse at night. Soon, an actual stiffness of the muscles of the legs begins to manifest itself, which becomes apparent principally when passive movements are attempted or even when the patient himself seeks to move his limbs. At last some amount of rigidity of muscles may be more or less continuously present, so as greatly to interfere with locomotion, or, in some cases, even to prevent it altogether.

In the early stages of the disease, ankle-clonus can be elicited with the greatest ease, and the knee-jerk is found to be distinctly exaggerated on both sides. When one of these patients is in the sitting posture, pressure commencing on the toes of one foot, as in the act of rising, will at once initiate the characteristic tremors of ankle-clonus. All such signs, however, will probably diminish as the rigidity becomes more marked.

Whilst the patient is able to walk he often exhibits a typical 'spastic gait.' The legs are generally kept close together, owing to a spasmodic contraction of the adductors of the thighs; the toes trail or are dragged along the ground; and then, when the heel is beginning to be brought down, a spasmodic contraction of the calf muscles may take place, tending to raise the patient upon his toes and almost throw him forward. In this way, a mixed and very irregular kind of progression is seen, partly to be accounted for by mere paresis, and partly by the occurrence of strong muscular spasms. In some instances, either owing to variations in the amount of the spasms or, it may be, to the great weight of the patient, this spastic gait is not well-marked. In all cases, however, it is quite different from the ataxic gait; and, when standing with feet close together, no increase of unsteadiness or feeling of vertigo is occasioned when the patient closes his eyes.

Sensibility is little, if at all, affected; still, in some instances it is apt to be slightly impaired. In one case, at present under the writer's care, ability to recognize differences of temperature was for a time greatly lessened; and although tactile sensibility is scarcely at all interfered with, the patient has frequently complained of a diminished power in appreciating the exact positions of his legs. Skin-reflexes are often normal, but occasionally they may be slightly increased.

The muscles do not atrophy, and their electrical reactions continue to be almost normal; while, according to Erb, that of the nerves is slightly but distinctly lowered to both currents. Sexual desires are not affected, but sexual disability may be occasioned to a variable extent—partly owing to weakness or actual paralysis, and partly to mere spasms of muscles. Micturition is often scarcely at all interfered with; there is nothing like incontinence of urine or of fæces, though there may be obstinate constipation, sometimes not continuous but rather of an intermitting type.

No vaso-motor or trophic disturbances in the limbs are usually present.

As the disease progresses (it may be very slowly, and in the course of years) the muscles of the trunk become affected, so that weakness and spasms (often of a very painful character) occur in the abdominal and back muscles. After a time the arms also may become implicated, and in the same fashion as the legs, excepting that when permanent contractions of the muscles come on, they mostly fix the arm to the side, though the forearm is pronated and semi-flexed, while the fingers and wrist are strongly flexed.

In rare cases the disease is limited to one side of the body, beginning, for instance, first in one leg, and then extending to the arm on the same side, so as to present a kind of hemiplegic distribution. Just as rarely, too, the disease may first affect both upper extremities, subsequently extending down the trunk, and ultimately involving the lower extremities.

During the development of the disease, shivering fits (affecting the muscles of the jaws as well as almost all the muscles of the body) may occur from time to time, lasting for half an hour or more; and, though quite unaccompanied by any changes of temperature, they may, nevertheless, be provoked by cold. Sometimes, however, such attacks of shivering occur spontaneously; or they may spread from some accidentally initiated ankle-clonus, or other well-marked spasm.

Persons suffering from this disease sometimes remain in an almost stationary condition, for a series of years, at any particular stage of the disease that may happen to have been attained. Ultimately, however, there is a tendency to complete paralysis of the parts affected, with permanent contractures—the legs at this stage being often immovably fixed in a condition of rigid extension. As a rule, pains are not complained of at any stage of the disease; still, some patients suffer much from painful cramp-like contractions, occurring either in the lower extremities, or else in some of the abdominal muscles.

So long as the morbid process remains limited to the lateral columns no other symptoms present themselves. Should it, however, invade the grey matter in particular regions of the cord, then characteristic complications are apt to arise, and it may also be said that the gravity of the disease becomes very distinctly increased. The way for a fatal termination may then be paved through the gradual increase, for instance, of bladder-troubles; or through the occurrence of severe bed-sores, and the collateral results to which they may give rise.

Another possible extension of the sclerosis is to the posterior columns, so that we may get a variable mixture of the symptoms pertaining to 'spasmodic spinal paralysis,' and to 'locomotor ataxy'. It should be borne in mind, however, that such a complicated clinical grouping sometimes develops in the reverse order.

Usually in patients suffering from this disease, there is no association with cerebral symptoms, nor is there any tendency to the springing up of cerebral complications. Still, in one case under the writer's care, a subacute maniacal condition became developed; while, in another case, diabetes to a slight, but tractable, extent has manifested itself. In both instances, however, there happens to have been a marked hereditary predisposition to the occurrence of insanity and of diabetes respectively.

In regard to **diagnosis** it should be said that the grouping of symptoms met with in this disease is so characteristic that there ought to be no difficulty in recognizing it. In no other affection of the spinal cord have we the combination of a gradually progressive paralysis beginning in the lower extremities associated with muscular twitchings and rigidities; greatly exalted tendon reflexes; no impairment of sensibility and no pains; no wasting of muscles or other trophic changes; and no interference with the functions of the bladder and rectum.

The real difficulty arises in the recognition of the complex forms of the disease, or of combinations of this disease with others then coming under observation for the first time. This, for instance, is the case where we have to do with a combination of posterior and lateral sclerosis, in which, in order to arrive at a diagnosis of the existing condition, the observer must be able to recognize the respective effects or modifications that may result from the combination of the two diseases. Another difficulty of the same kind arises when the symptoms of the disease are complicated by extension of the sclerosis to the grey anterior horns, the characters of which condition are now about to be referred to under the head of 'amyotrophic lateral sclerosis.'

Again, when 'multiple sclerosis' affects in the main the lateral columns, the real diagnosis can only be arrived at by the recognition of symptoms which could not be produced by a mere affection of the lateral columns.

25. AMYOTROPHIC LATERAL SCLEROSIS.

This is an extremely interesting and rare affection which might perhaps be regarded as a mere variety of 'primary lateral sclerosis'; still, it is a variety which pursues a very distinctive course, and constitutes a disease much more formidable than its prototype, since it seems almost invariably to lead to a fatal termination in two or three years.

The peculiarity of this form of lateral sclerosis lies principally in the fact that it commences in the cervical region, and soon spreads to the contiguous anterior horns of grey matter; thence, after more or less of an interval, it extends in two directions :—(*a*) downwards, so as to involve the dorsal and lumbar lateral columns, and also the contiguous anterior cornua of grey matter; and (*b*) upwards, so as to implicate the upper cervical region of the cord and the bulb in a similar fashion.

Thus, it will be seen that there are three peculiarities about this form of lateral sclerosis; (1) that it begins in the cervical region of the cord, and subsequently affects the lumbar portion; (2) that it does not remain limited to the lateral columns, but soon spreads to the contiguous anterior cornua, where it leads to destruction of the great motor ganglion-cells; and (3) that it almost invariably extends upwards also, so as to involve the bulb, and thus gradually brings about the death of the patient.

Being marked by such anatomical characters as have been above enumerated, it will be easily understood that patients suffering from this disease present an admixture of such signs and symptoms as may be met with separately in 'progressive muscular atrophy,' in 'lateral sclerosis,' and in 'progressive bulbar paralysis.' We have, in fact, the following typical grouping and sequence of symptoms :—

1. Paresis, gradually increasing to actual paralysis of the upper extremities, and soon associated with distinct atrophy, and fibrillar twitchings of muscles. Any movements that can be executed are weak, and associated with tremors. More or less marked rigidity of muscles, and, finally, actual contractures occur, in which the arms are fixed close to the sides of the body, the forearms being semi-flexed and pronated, while the hands and fingers are strongly flexed.

2. After an interval of some months, a similar group of symptoms becomes developed in the lower extremities. Again, we have paresis gradually increasing, with muscular tensions, exaggerated tendon-reflexes, and an increasing amount of rigidity of the lower limbs which are usually fixed in the extended position. At a later period in the lower extremities, as compared with the arms, a process of muscular atrophy sets in, with or without development of the 'reaction of degeneration,' and fibrillar twitchings in the affected muscles.

During the whole of this time, there is little or no interference with sensibility. There is usually no implication of the sphincters, and no tendency to the formation of bed-sores.

3. In the last stage of the disease, there is evidence of extension of the morbid process upwards to the upper cervical region and the medulla. Signs of bulbar paralysis present themselves in the usual way, by paralysis with atrophy of the tongue and lips, and by progressive weakening of the muscles of the palate, pharynx, and larynx. The phrenic nerve has also generally become involved, and when weakness of the diaphragm is added to weakness or actual paralysis of the other muscles of respiration, this all-important function becomes more and more impaired, and thus a fatal termination may at any time be easily brought about. Increasing difficulty of articulation and deglutition may have existed for some months before death.

From the point of view of **diagnosis**, it should be said that in the early stages, when amyotrophic lateral sclerosis affects the arms only, it is characterised by its gradual, painless onset, the absence of impairment of sensibility, the fact that weakness sets in first, and that twitchings and tensions of muscles soon declare themselves, either before or after the muscular atrophy becomes very obvious. This combination is already sufficiently distinctive in the absence of pain in the back, tenderness over the spine, or any other evidence of vertebral disease.

When the disease advances to its second and third stages, the clinical picture becomes gradually more and more distinctive, and easily to be separated from that presented by all other affections of the spinal cord : especially if we are duly impressed by the negative symptoms, namely, the absence of sensory impairment, of bladder-troubles, and of bed-sores.

26. LOCOMOTOR ATAXY.

This is a disease which occurs most frequently between the twentieth and the fiftieth year. It is much more common in males than in females. It is often called 'tabes dorsalis' or 'posterior spinal sclerosis.'

It is a disease concerning which much might be said both in regard to etiology, and also in regard to the excessively wide range of symptoms that may at times be included under it, in one or other patient; but we cannot pretend to enter into these discussions or descriptions here. We shall limit ourselves to such a brief outline of the symptomatology of the disease as may be needful for the proper understanding of the points upon which its diagnosis depends.

The patients in whom locomotor ataxy develops have often a distinctly neurotic history. The exciting causes are generally extremely obscure. Marked sexual excesses seem occasionally to be connected with its development. Syphilis is, in my opinion, greatly overrated as a cause of this disease. I have seen numbers of cases in which the most careful examination and enquiry have failed to reveal any evidence of pre-existing syphilis. I believe that it has not existed in one-fourth of the numerous cases of locomotor ataxy that have come under my observation and treatment.

The disease is dependent, in the main, upon a sclerosis of the posterior columns of the cord in the lumbar region, whence the process extends upwards so as to involve the same parts in the dorsal and in the cervical regions. It has been shown by Pierret that it is the implication of the posterior root-zones in this process which seems to give rise to the symptoms of the disease. Strumpell, however, has recorded two or three cases which seem opposed to this view; so that it cannot be considered to be, as yet, definitely settled. It is true that the process of sclerosis often extends so as to involve the whole or the greater portion of the posterior columns of the cord. As Lockhart Clarke pointed out, it also occasionally extends so as to involve the posterior cornua of the cord.

Again, it is right to mention that Déjérine has recorded a few very important cases in which there were symptoms closely resembling those met with in locomotor ataxy, but where no sclerosis of the posterior columns existed—the symptoms being, in these cases apparently produced by a neuritis affecting many of the peripheral nerves. It will be for future investigations to show, now that attention has been drawn to the subject, what is the relative frequency of such affections (which are possibly quite exceptional); and also what are the most important points by means of which the diagnosis may be made in these exceptional cases.

The disease is eminently chronic. It may develop very slowly; and certainly the individuals affected may live for long periods—ten, twenty, or even thirty years.

In different cases of locomotor ataxy, there is often considerable variation in the rapidity of evolution, as well as in the order of evolution, of the different symptoms.

It will be convenient to divide the signs and symptoms into three categories—viz. (1) those of the early stages; (2) those of the established disease; and (3) occasional signs and symptoms.

(1).—Among the early symptoms of locomotor ataxy about the most constant are the peculiar lightning pains in some parts of the lower extremities (often believed to be rheumatic or neuralgic in character). These pains may precede other symptoms by months or years.

Some amount of diplopia, due to paralysis or paresis of one of the muscles of the eyeball—usually of a temporary character—with or without slight ptosis.

Loss of knee jerk on both sides.

Occasional difficulty in voiding or in holding urine.

Sometimes a distinct increase of sexual desire and ability.

(2).—As additional symptoms of the established disease the following may be enumerated:—

Difficulty of standing or walking with the eyes closed, or when in the dark. It may be first noticed by the patient while washing his face in the morning; but is always easily recognizable by making him stand before us with his feet close together, first of all with eyes open and then with eyes closed, the latter condition usually at once causing the patient either to reel or show some slighter signs of unsteadiness.

The pupils are often notably contracted and insensitive to light; if they are not so much contracted, or if they are of medium size, they are usually either insensitive or very sluggish to light, although they contract well to accommodation—a combination of characters often spoken of as the 'Argyll-Robertson phenomenon.'

The patient exhibits a more or less distinct ataxic gait, the heel being commonly brought to the ground first—often with a stamp—and the legs moved about in a more or less irregular or spasmodic fashion. It is often needful for the patient to look at his feet, or at the ground just in front, while he is walking. Similarly, when lying in bed, if one foot and leg be raised off the bed, it cannot be kept steady but sways about in a more or less marked manner. And when in the same position, if the eyes be shut and the feet placed far apart, the patient cannot readily raise the one foot and bring its heel down upon the great toe of the other foot—he generally goes more or less wide of the mark.

When the upper extremities are also affected it is found that the patient's ability to pick up a small object, such as a pin (or, in more advanced cases, even a pencil), from a smooth surface is greatly interfered with. He does it with difficulty, or, in an altogether irregular manner. Or, again, if his arms be extended at right angles with his body, and he then attempts, with eyes closed, to bring the forefinger of either hand rapidly to the tip of his nose, it will be found that he goes more or less wide of the mark and only brings his finger into the desired position after some wandering.

Sensibility is interfered with in various ways, first in the lower extremities, afterwards in the trunk and upper extremities. Beyond the pains already referred to, numbness may be felt in the lower extremities, or occasionally there may be a girdle sensation round the waist; the patient also derives unnatural sensations from the soles of his feet when standing or walking—which are either wrong in kind or merely defective in amount (he seems to be treading on cotton wool, or, at all events, not to feel the ground adequately). These are the anomalies of sensibility of which he may complain spontaneously.

On careful examination, however, it will generally be found that sensibility is affected in various modes. Thus the kinæsthetic sense is generally more or less defective, so that after passive movements of the limbs in different directions, when the patient's eyes are closed, he cannot at the end correctly say in what position such limbs are left. Again, when he awakes from sleep he may not be able to say correctly (before he moves them, and thus gets information through the sense of touch) what is their exact position in the bed. His ability to discriminate differences in degree of muscular tension (estimation of weights) is also defective.

Tactile sensation is also blunted, delayed, wrongly localised, or more or less lost in various parts of the lower extremities or trunk. Plantar anæsthesia is often well marked; in other parts the anæsthesia is apt to be patchy and irregularly distributed. More rarely, it is mixed up with areas of hyperæsthesia, or with areas in which the slightest touch gives rise to most marked reflex movements of the lower extremities.

Sensibility to painful impressions and to differences in temperature may also be variously interfered with in different parts of the body; though such defects, like those of other modes of sensibility, are generally most marked in the lower extremities.

The special senses are far less uniformly impaired. Sight is most commonly affected; hearing only occasionally; and smell still less frequently. The defect of sight (due to atrophy of the optic nerves) sometimes shows itself very early, and at other times only at later stages of the disease—in any case it may go on to the establishment of a complete amaurosis.

In males, erections become less frequent and often cease altogether, sexual desire concurrently growing less and at last disappearing. Exceptions to this rule are rare, though they are occasionally met with.

(3).—Occasional signs and symptoms in Locomotor Ataxy. These are of various kinds, but the principal of them are the so-called 'gastric crises' and various trophic troubles—such as skin eruptions, perforating ulcers, arthropathies, and muscular atrophies. It will only be needful to say a few words concerning each of these signs.

The term 'gastric crises' has been applied to irregularly periodical attacks of pain at the epigastrium associated with vomiting, headache, and occasionally diarrhœa, generally without elevation of temperature. Such attacks may last for hours or days, and then disappear completely for an altogether variable interval.

Sometimes in cases where the lancinating pains are very severe, eruptions of herpetic vesicles have appeared, from time to time, in the regions of skin to which such pains are referred. (This sign may be fairly considered to indicate the existence of some amount of peripheral neuritis.)

Occasionally deep perforating ulcers are met with in the soles of the feet even in the early stages of locomotor ataxy, which seem to come on without any adequate exciting cause. (This also may, perhaps, be a sign related to a peripheral neuritis.)

Peculiar joint affections ('Charcot's arthropathy') in which the changes that ultimately arise are closely related to those of rheumatoid arthritis, are sometimes met with in association with locomotor ataxy. Such changes affect most frequently the knees and the ankles, less frequently the elbows and the hips. They show themselves sometimes in the early stages of the disease, but more frequently when it is well advanced. The changes begin somewhat abruptly with pale œdematous and painless swellings of the joints affected. In later stages, that is, after the joint affection has lasted some years, there commonly ensues great deformity of the heads of the bones, with atrophy and much destruction of ligaments, leading, not unfrequently, to dislocations of an incurable character. (Charcot believes that these joint changes or arthropathies are due to extensions of the morbid process from the posterior columns so as to invade some portions (the precise locality of which cannot at present be defined) of the anterior cornua of the cord. The writer, on the contrary, thinks they are much more likely to be due to morbid changes in the posterior nerve roots or in the peripheral nerves of or from which the joints are innervated.)

Muscular atrophies occasionally occur in the later stages of locomotor ataxy, principally in the muscles of the lower extremities. As such changes advance the ataxy becomes associated with more or less of actual paresis, gradually deepening to paralysis. Autopsies in such cases have shown that the process of sclerosis has extended from the posterior columns so as to involve the groups of large cells in the anterior cornua and cause them to become atrophied. (There is, therefore, the strongest reason for believing that the muscular atrophy sometimes associated with locomotor ataxy, is due to an extension of the morbid process from the posterior columns to the anterior cornua of the cord.)

Occasional association of Locomotor Ataxy with other diseases of the Spinal Cord and Brain.—The associations which are most likely to be met with are one or other of these five, viz., with lateral sclerosis of the spinal cord; with general paralysis of the insane; with progressive muscular atrophy (or Cruveilhier's palsy); with ophthalmoplegia externa; or with hemiplegia.

In certain cases disease in the posterior columns of the cord is followed by similar changes in the lateral columns, or the reverse order of development may be met with (see p. 638). These are mostly cases of disseminated sclerosis of 'spinal type'; and when they exist, we get an admixture of the signs peculiar to posterior and to lateral sclerosis respectively.

General paralysis of the insane occasionally develops in a patient already suffering from locomotor ataxy. This happened recently in one of my patients who had been under observation from time to time for the previous six or seven years, having been originally treated by me for locomotor ataxy in its early stages. The progress of this disease was subsequently arrested, and for three or four years before the outbreak of the symptoms of general paralysis he had been suffering comparatively little from the locomotor ataxy. In another case also, which came under my treatment only in the early stage of general paralysis of the insane, which proved rapidly fatal, I understood from the patient's medical attendant that locomotor ataxy had been diagnosed about eighteen months previously by an eminent physician. Seeing that in locomotor ataxy itself there are no signs of mental disorder, the supervention of general paralysis of the insane with its 'delusions of grandeur' and generally self-satisfied condition, is marked off by a well-defined change in the symptomatology.

The combination of locomotor ataxy with Cruveilhier's palsy, also well developed, is a very rare combination of which I have only seen one example. The case was sent to me by the patient's medical attendant simply as one of locomotor ataxy, and as he entered my consulting room I was much struck by the ataxic gait in combination with a typical flail-like aspect about both upper extremities. On examination, great atrophy of the pectorals and shoulder muscles was found on both sides, as well as of nearly all the muscles of the upper extremities. The locomotor ataxy had existed for about four years, and the arms had not been appreciably affected till the atrophic process set in, and this had existed for about twelve months before he came under my observation.

Ophthalmoplegia externa is decidedly more common as an affection coincident with locomotor ataxy. I have myself only seen one case, but several have been recorded by Buzzard and by Hutchinson. The latter observer thinks such a combination is most apt to occur when syphilis has pre-existed as an etiological factor.

The coexistence of hemiplegia with locomotor ataxy is one which occurs every now and then. This is scarcely to be wondered at seeing that locomotor ataxy is such a chronic disease, and that those who suffer from it are, of course, still liable to the occurrence of ruptures or thrombotic occlusions of one or other of their cerebral arteries. In this class of case, therefore, we have to do with a more purely accidental association than that which exists in either of the other combinations above referred to.

We have hitherto spoken of locomotor ataxy in such a way that it might be inferred that it is generally a 'progressive' disease. This, however, is by no means uniformly the case; it is often extremely irregular in the mode of its development. Thus, the characteristic pains may precede other obvious symptoms in some cases for five or even ten years; or, the disease may stop short at some other initial combination for a long term of years—Buzzard speaks of one case in which the only symptoms were the existence of lightning pains, absence of knee-jerk, and blindness due to white atrophy of the optic discs. Again, in cases where the disease is more completely evolved, the morbid process may at any time cease to make further progress, so that such a patient may remain for long periods in practically the same condition.

27. INSULAR OR DISSEMINATED SCLEROSIS OF THE SPINAL CORD.

This is a disease produced by the development of patches of sclerosis (overgrowths of neuroglia) of varying size and shape, throughout the spinal cord, and often also, at the same time, in different parts of the brain.

Clinically the disease is met with under the most diverse forms, according to the different sites and sizes of the patches of sclerosis occurring in different cases. These various forms of the disease are divisible into three partially distinct types, according as the morbid changes and symptoms occur in and are referable to (1) the spinal cord alone (*spinal type*); (2) to the cerebrum alone (*cerebral type*); or (3) to the brain and spinal cord (*cerebro-spinal type*). As the dominant symptoms of the disease are often those of the spinal type, even where there is also an extension of the morbid process to the cerebrum, it will be most convenient to speak here in the main of the 'cerebro-spinal' type. This is, moreover, both more frequent and a more characteristic malady than either of the simpler forms of the disease.

In regard to the ætiology of this affection, little can be said. It may occur with or without the predisposing influence of a neurotic tendency. It is at least as common in females as it is in males; and though rarely occurring in children under ten years of age, it is perhaps most common between the ages of ten and thirty years. Beyond the age of forty, it again becomes very rare.

Among the exciting causes, exposure to wet and cold would seem to take the first rank. After this come traumatic influences of various kinds, mental shocks or troubles, great fatigues from mental or bodily labour, and finally the state of convalescence from several acute diseases, such as typhus, cholera, variola, or other specific fevers. It has, indeed, been said to occur sometimes as a sequence to severe and long-continued hysteria; but in some of such cases at least it would seem to be far more probable that the early and obscure symptoms connected with this affection were those which were regarded as hysterical. 'Hysteria' may be produced or simulated in many ways, but, as itself a producer of organic changes in the spinal cord, its *rôle* is assuredly open to grave doubts.

It can easily be understood, from what has already been said, how much the symptomatology of this disease is liable to vary in different cases, according to the varying situation, extent, and order of evolution of the morbid patches. That it is possible to assign anything like a definite symptomatology for the affection is due to the fact that there are certain seats of election, in which the patches of sclerosis are specially apt to occur. The sites affected with special frequency are the lateral columns of the spinal cord, the bulb, and the pons; and it is with the occurrence of patches of sclerosis in these situations that we have the following set of correlated symptoms pertaining to the 'cerebro-spinal' type of the disease.

A slowly ensuing paresis of the lower extremities begins, first in one limb and then, after a variable time, in the other. During this period, the paresis develops into a more and more marked paralysis, though the sensibility of the limbs remains almost completely unaffected—nothing more than a temporary numbness being complained of in the majority of cases, while lightning-like pains and girdle-sensations are generally altogether absent. After an interval, first one and then another upper extremity may become weak, and subsequently more or less paralysed. During these early stages of the disease more or less distinct remissions of symptoms are apt to occur from time to time.

Meanwhile, a most typical sign soon shows itself in the paretic or semi-paralysed limbs, in the form of a marked trembling or shaking of those muscles or parts of a limb which are called into voluntary action with any intensity, although these phenomena immediately subside when the voluntary exertion ceases. The involuntary movements consist either of extremely well-marked tremors, like those met with in some cases of paralysis agitans, or else of movements of greater range, more resembling those of chorea.

Later, some paresis of the trunk-muscles may occur, as well as of those of the neck; and this may be followed by a similar affection of the tongue, lips, and facial muscles—possibly, also, of those of the palate, pharynx, and larynx. When a patient affected in this manner, who has been previously lying perfectly still in bed, is told to endeavour to sit up, shakings and tremors begin in almost all parts of the body, and the scene is strangely changed until all voluntary efforts cease, and the recumbent position is again assumed. The same kind of thing is seen when movements of particular parts of the body are attempted: thus when, in the sitting posture, the patient attempts to hold up one leg, tremors of it immediately begin; ask him to take hold of something or to squeeze a dynamometer, and the upper extremity called into action at once begins to shake; request him to put out his tongue, and immediately irregular protrusions of the organ occur, associated with twitchings about the angles of the mouth and even in other parts of the body. The act of walking may cause, in more or less advanced cases, tremors of the legs, arms, trunk, head and neck—all at the same time.

Movements of slight intensity occasion either no shakings or merely tremors of a very fine kind. The latter are seen in the early stages of the disease, when writing is attempted. Almost each letter registers a number of fine tremors, mixed here and there with greater irregularities. In more advanced cases, however, the movements are so disorderly that writing becomes either impossible or wholly illegible.

Just as there is no loss of ordinary sensibility, so we find that patients generally remain fully conscious as to the positions and movements of their limbs, and that closure of the eyes occasions no increased uncertainty of their movements; nor, when in the standing position, are they rendered more giddy or more unsteady by such a proceeding. All these signs may, however, be different in those cases in which the posterior as well as the lateral columns of the cord are distinctly affected. Such cases are met with not unfrequently.

Up to this stage, there may be no distinct interference with the functions of the bladder or the rectum. The tendon-reflexes are, however, generally distinctly exaggerated; ankle-clonus may be obtained with readiness, and the knee-jerk is often more pronounced than usual. There is no tendency to the formation of bed-sores; no wasting of muscles; nor is any alteration in their electrical excitability met with.

After variable and often long periods, the affected lower extremities, which have become more and more paralysed, may, in some cases, show signs of commencing bar-like rigidity. The limbs, as the patient lies in bed, are closely drawn together, and in a condition of rigid extension, which is generally increased when any attempts to move them are made. At first this condition of the limbs ensues from time to time, in the form of paroxysms lasting for an hour or two. But, after a period, the attacks are both more frequent and longer, so that ultimately the condition of rigidity becomes permanent. Contractions of the arms are less common, and when they occur they become fixed at times in a different position from that met with in simple lateral sclerosis (p. 637), that is, like the lower extremities, in a condition of extension, and closely drawn to the sides of the body.

At this period ankle-clonus can often be elicited with the greatest ease, and the movements of the one leg may extend to the opposite lower extremity, and, indeed, set up more or less of general tremor throughout the body. Exposure to cold, or irritation of the skin in various ways, will also often suffice to set up the same kind of general tremor.

The manifestation of tremors of the tongue, lips, and face is, of course, a sign that the bulb is affected; and when this occurs, simultaneously or very soon after, other evidences of implication of the bulb and of contiguous portions of the cerebrum may be expected to present themselves. Articulation may become more or less affected, the speech being rendered slow, hesitating, and measured, syllable by syllable (of 'scanning' type); or it may be jerky in character—becoming especially thick and blurred in the later stages of the disease. The power of swallowing is less frequently impaired, but in advanced stages it is apt to be affected.

Nystagmus is very frequently met with. Diplopia, or actual paralysis of the ocular muscles, is rare. Amblyopia not unfrequently exists; perhaps in one eye only. Actual blindness is very rare.*

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* In support or rather in explanation of this, there is the fact mentioned by Charcot, that an optic nerve which was affected through its whole thickness by a patch of sclerosis was yet capable of performing its functions.

Vertigo, sometimes to a marked extent, is no uncommon symptom; and, as the cerebrum becomes more and more affected, a condition of well-marked hebetude, or actual dementia, gradually becomes pronounced. This betrays itself externally by a blank, expressionless aspect of the face; the patient becomes childish in manner, his memory fails, he takes interest only in trifles, laughs constantly also at the merest trifles, or, on the other hand, is very easily moved to tears.

During this condition of things a subacute maniacal condition may supervene; or the patient may develop 'delusions of grandeur' precisely similar to those met with in 'general paralysis of the insane'—examples of which the writer has recently seen in two of his own patients. In other cases, persons suffering from this disease may lapse into a profoundly melancholic condition.

At this stage, too, apoplectiform or epileptiform attacks are particularly apt to occur from time to time. After such attacks, of whichever kind, the limbs on one side of the body and the face are left more or less paralysed; and where the attack has been epileptiform in character, the convulsive twitchings are often limited to this one side of the body. As Charcot has pointed out, these attacks are precisely similar to those that occur in general paralytics, or in cases of old hemiplegia with descending sclerosis. They answer to the so-called 'congestive attacks,' but, as Charcot contends, they do not seem to be associated with any new appreciable lesions of a 'gross' order. Such epileptiform attacks may be brief, or they may last for hours; or, off and on, even for days. In all of them the temperature begins to rise almost at once—without any initial period of depression—and may even reach 104° F. in a few hours, or otherwise more slowly. The temperature then begins to fall again; or, should it continue to rise to a still higher point, the attack is very apt to terminate fatally.

Every attack of this kind leaves the patient in a manifestly worse condition, both bodily and mentally; and perhaps in one of them, at last, death may occur.

The symptomatology of this disease is liable to be considerably modified in different cases, but principally in two directions, producing complications of the same kind as those that are also apt to occur in 'spasmodic spinal paralysis.' In each disease there may, in some cases, be a special affection of the posterior columns, in one or other region of the cord, bringing with it more interference with sensibility, and an admixture of other symptoms pertaining to locomotor ataxy. It is, perhaps, principally in these cases that the '*crises gastriques*' (pains, vomiting, and occasionally diarrhœa) are also met with. In other instances, there may be an extension of the sclerosis to the grey matter of the anterior cornua in one or other region (as well as to other parts of the grey matter), leading, among other phenomena, to muscular atrophy in related regions of the body. In either of these ways, the symptoms of the original disease may be complicated, and, to a certain extent, obscured.

Many other differences also present themselves in special cases, owing to the varying situations in which the morbid patches make their first appearance. In a fair proportion of the cases, the disease seems to reveal itself first in the brain, rather than in the spinal cord.

After pursuing a very slow course for years (often five to ten), the unfortunate sufferers from this disease may at last be carried off in various ways. Death may take place in one of the apoplectiform or epileptiform attacks, occurring either in patients who are merely slightly demented, or in those who are otherwise actually insane; or, at last, in cases in which there is great interference not only with articulation but also with deglutition, the functions of the heart or of respiration may also become affected, and may thus lead on to a fatal termination.

In other cases, after the disease has lasted for years, and when the grey matter of the cord has become seriously involved, accidents may supervene similar to those which occur in the final stages of many cases of paraplegia. The bladder may become paralysed, and after a time inflammation and ulceration may be set up, followed by secondary inflammation of the ureters or kidneys. Or, bed-sores may form; sloughing may go on extensively; and the patient may at last die exhausted, or from the supervention of blood poisoning or some acute inflammatory disease.

In its early stages the **diagnosis** of this disease may present very considerable difficulties. This is especially the case when the morbid process begins in the cerebrum. Here, for a time, there may be nothing distinctive, and we have to wait for the further development of the disease before anything like a positive diagnosis is possible. Similarly, where the disease begins only with spinal symptoms, it is often extremely difficult to diagnose it with certainty in its very early stages. The important characters in the more typical forms of the disease are the youth of the patient, the paresis gradually increasing first in one and then in the other lower extremity, with no alteration in sensibility or in the electrical irritability of the nerves or muscles. When ankle-clonus becomes easily obtainable, and when, moreover, the peculiar tremors and disordered movements on voluntary excitation of the muscles are met with, together with the absence of any such tremors in the condition of rest, and some amount of paresis or of similar symptoms in one or both upper extremities, the diagnosis of the 'spinal' type of this disease can be no longer difficult or doubtful.

By far the most typical cases, however, are those of the 'cerebro-spinal' type, in which, with such symptoms as are above indicated, there are also some others due to disease of the bulb or pons—such as have already been indicated in speaking of the symptomatology of the disease. In these cases the disease is really quite distinctive; so that, even when the patient is seen at this stage for the first time, the malady ought to be easily recognized.

Still such cases are, with extreme frequency, supposed to be instances of 'hysteria.' Chorea is another affection with which it is most apt to be confounded; but the absolute cessation of all tremors and disordered movements in multiple sclerosis when the patient is at rest, and their immediate re-initiation (mainly in the parts moved, but also often to some extent in others) on the occurrence of voluntary efforts, is a thoroughly distinctive characteristic.

Paralysis agitans ought to be distinguished from disseminated sclerosis with even more ease. It is scarcely ever met with in persons under the age of thirty-five, just as multiple sclerosis is only rarely met with in persons beyond such an age, the movements of paralysis agitans are only to a slight extent exaggerated by voluntary exertion of the parts, or they may even be stopped thereby. Nor do the movements met with in paralysis agitans, in the form of fine tremors, cease to anything like the same extent under conditions of rest. There is generally no shaking of the head and neck in paralysis agitans, though they are inclined forwards, in association with a stooping gait, in a characteristic manner.

Mercurial poisoning, with tremors, can be easily distinguished, on inquiry into the history of the patient, and the mode of onset of the disease.

In those more irregular cases of multiple sclerosis, in which there is either an implication of the posterior columns of the cord, or of the sibility, and an admixture of other symptoms pertaining to locomotor ataxy. It is, perhaps, principally in these cases that the '*crises gastriques*' (pains, vomiting, and occasionally diarrhœa) are also met with. In other instances, there may be an extension of the sclerosis to the grey matter of the anterior cornua in one or other region (as well as to other parts of the grey matter), leading, among other phenomena, to muscular atrophy in related regions of the body. In either of these ways, the symptoms of the original disease may be complicated, and, to a certain extent, obscured.

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In those more irregular cases of multiple sclerosis, in which there is either an implication of the posterior columns of the cord, or of the grey matter in some region or regions, the diagnosis of the complex nature of the affection must be based upon the general principles applicable to the regional diagnosis of spinal cord disease.

28. FRIEDREICH'S DISEASE.

This is a disease apt to show itself in several members of the same family, at periods varying mostly between early childhood and the twenty-fifth year. It has been found to be two or three times more frequent in males than in females. It is not an hereditary disease in the strict sense of the term ; it is rather a family disease, showing itself in several children of the same parents, although neither they nor their parents may have had the disease. The term 'Hereditary Ataxy' is therefore misleading.

The disease is dependent upon the development of areas of sclerosis in the posterior and in the antero-lateral columns of the cord, as well as in some parts of the grey matter—together with an extension of such morbid processes to the bulb, so as to implicate the hypoglossal nuclei and other parts. This affection has, therefore, likewise been named 'Diffuse Sclerosis of the Spinal Cord and Bulb.'

Commencing without any special exciting cause, the malady shows itself first by the occurrence of a gradually progressive weakness in one or both lower extremities, together with an unsteady or uncertain, rather than a distinctly ataxic gait. The legs are often placed wide apart, and the walk is not unlike that of a drunken man. By slow degrees the weakness increases, till even standing becomes impossible.

The weakness and uncertainty of movement soon extends to the upper extremities, which also become tremulous during the execution of movements. Most frequently this inco-ordination is not exaggerated by closure of the eyes.

Later on the trunk and the head become the seat of oscillations which are exaggerated when the patient executes any movement, but which, on the other hand, cease when he is completely at rest in the recumbent position.

In males a condition of impotence supervenes ; and in females the menstruation becomes exceedingly irregular. These signs are not long in showing themselves when we have to do with patients past the age of puberty.

After some years, the tongue partakes in the trembling; and speech becomes hesitating, thick, and at last scarcely intelligible. Ultimately the tongue may become completely paralysed and motionless.

Nystagmus of a slow horizontal character is apt to show itself, on the occasion of voluntary movements of the eyeballs.

During the final stages of the disease, there is apt to be more or less complete paralysis of all the limbs—often associated with muscular atrophy. At this period, also, the limbs are occasionally affected with cramps, or transitory contractures.

Again, it is only during the final stages of the disease that affections of sensibility are prone to show themselves—and that, principally, in the form of anæsthesia of the lower extremities, often so complete as to involve the joints and muscles as well as the skin. Sometimes pains are complained of, but they are rather erratic in character than of the lightning-like or lancinating type.

The special senses and the intellect usually remain intact.

The pupil often remains sensitive to light, and may be even rather unnaturally sensitive.

The sphincters are not affected; and there is no tendency for bedsores to occur.

Sudden attacks of vertigo are apt to supervene (and that quite irrespective of the position of the patient at the time); or towards the close of the disease actual apoplectiform attacks may occur, in one of which the patient may succumb. Such an attack is said to be of the following type:—"It is characterised by a rapid but incomplete loss of consciousness, by resolution of all the limbs and a generalised anæsthesia, by a considerably impeded respiration which is of a jerking and noisy type, by a tumultuous action of the heart and great frequency of pulse (130), together with a notable elevation of temperature" (Brousse).

The progress of the disease is often extremely slow, but always fatally progressive. It may last from 5-30 years.

When first recognized this disease was regarded as a form of 'locomotor ataxy'; but, as Charcot and Bourneville have pointed out, some of the cases at least are much more closely related to 'disseminated sclerosis.' Friedreich's disease is, in fact, a malady as it were intermediate between these two affections, having important alliances with each of them, though, for the most part, the alliance is closer between it and disseminated sclerosis than between it and locomotor ataxy.

As already indicated, the diagnosis has to be made principally between Friedreich's disease and disseminated sclerosis, or else between it and locomotor ataxy. Such difficulties of diagnosis must, however, present themselves principally in regard to the first case of Friedreich's disease that happens to show itself in a family. If the medical attendant is aware that already one or more of the patient's brothers and sisters has become affected in a similar manner, more than half the difficulty of diagnosis is at once got rid of—supposing the practitioner to be aware of the existence and general nature of such a malady as Friedreich's disease. This is so, because several cases of ordinary locomotor ataxy or of disseminated sclerosis in the same family must be regarded as events of extreme rarity.

LOCOMOTOR ATAXY.	FRIEDREICH'S DISEASE.	DISSEMINATED SCLEROSIS.
Rarely before 20th year.	Commonly before 20th year.	Often before 20th year.
Affections of sensibility early and constant. Sight and hearing often affected.	Affections of sensibility absent.	Affections of sensibility generally absent, though sight and hearing are sometimes affected.
Double vision, and Argyll-Robertson pupil present.	Double vision, and Argyll-Robertson pupil absent.	Double vision frequent, and Argyll-Robertson pupil absent.
No nystagmus.	Nystagmus frequent.	Nystagmus very common.
No oscillations of head and trunk.	Oscillations of head and trunk very common.	Oscillations of head and trunk frequent.
Knee-jerk absent; no ankle clonus. No rigidities.	Knee-jerk either absent, of medium intensity, or exaggerated. Sometimes clonus. No rigidities.	Knee-jerk commonly exaggerated, and clonus present. Rigidities common.
No speech defects.	Difficulties in speech.	Difficulties in speech.
Simple inco-ordination in arms common.	Tremors of arms exaggerated by movement.	Tremors of arms, on movement, gradually becoming choreic in range.
Mental disturbance rare.	No mental disturbance.	Mental disturbance frequent.

It should, however, be borne in mind that in Friedreich's disease (as well as in locomotor ataxy, and disseminated sclerosis) much variation exists in different cases, in the rapidity and in the order of development of the several signs and symptoms. A similar variability exists in regard to the relative development of these several signs, in different cases—the result being that only a generic similarity is likely to exist between any two or three consecutive cases of this disease that may chance to present themselves to the same observer.

Supposing, however, that we have to do with a solitary case of disease, then the question of age becomes of first importance. If the patient should be under twenty years of age, the chances would be decidedly against locomotor ataxy, and in favour of the case being either one of Friedreich's disease or of disseminated sclerosis. While the indications would be still further against locomotor ataxy, if there should be an absence of the peculiar pains characterising this latter disease, or of other affections of sensibility.

The points, in detail, which must be taken into consideration for the diagnosis of these three diseases from one another are given above in parallel columns.

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